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CASE REPORT NO. TF 17/1

VETERINARY/BOWEL OEDEMA

DATE: 9.6.60

ANIMAL: 16 piglets 10 weeks old (two litters)

SYMPTOMS: One pig, the "runt" of the litter had died with "vague" symptoms on 8th June. A second pig was found dead on 9th June after showing hind leg inco-ordination and a puffiness around the eye-lids.

At p.m. the mesentery and wall of the spiral colon were grossly thickened and oedematous, the stomach wall was oedematous.

DIAGNOSIS:
Bowel Oedema.

TREATMENT:
Remaining 14 piglets were each given 50 mg. Vetidrex i.m. (one of these piglets was showing hind leg inco-ordination) Mag. sulph. was administered simultaneously.

RESULTS:
Re-examination on the 11th June showed the piglet previously described as suffering from posterior inco-ordination to be normal and no further cases have occurred in the remainder of the litter.

ASSESSMENT:
The response was satisfactory.

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THE VETERINARY RECORD

Founded by William Hunting, F.R.C.V.S., in 1888

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The Medical Profession and Ourselves

THERE has always been a degree of collaboration between the sister professions of human and veterinary medicine, and the histories of these closely related sciences contain many examples of the gains to be obtained from this. The early records of the Royal Veterinary College show the great interest members of the medical profession took in helping to start the first centre of veterinary education in this country, and the recent opening in Glasgow of the Wellcome Laboratory for Comparative Studies has demonstrated once more that there exists to-day a strong feeling that such inter-professional co-operation must be fostered still further. Similar collaboration is going on in many of our universities.

The Loveday Committee made a very wise decision when it advised that the Veterinary Schools should be allied with the medical rather than with the agricultural Departments in Universities. Despite the many associations we have in practice with agriculture, the separate disciplines which are represented in the individual Departments in our Schools, and hence the subjects which our students are taught, have much more in common with the type of organisation found in medical teaching schools. One great danger has to be guarded against in this respect: the fusion of Departments so that both medical and veterinary students are taught by one staff. A certain amount of such pooling can be tolerated during the period in which the Schools are being built up to their final and proper academic status, and indeed it may be necessary in certain cases on financial and staffing grounds. But we are a relatively small profession, and we depend very largely for our research work and teaching advances on a fairly small number of people. No matter where this work is done—in our several excellent research institutes, in the universities themselves, in Government departments or in industrial laboratories—at least part, and possibly in the future an even greater part than at present, of the training of specialists has to be done in university Departments. This separate professional identity, with unity of interest, can be maintained only by having independent Departments in the universities, and by resisting the many arguments (some of which have obvious merits) which are put forward for the amalgamation of veterinary and medical Departments and the possible submergence of the former in the latter. It should never be forgotten that both human and veterinary medicine are, in large measure applied sciences, and that those who direct policy in both the under-graduate and post-graduate fields must be fully conversant with the whole picture.

This is not to suggest that we should not employ non-veterinarians in our Schools: far from it. Many workers from other disciplines have supplied, and will continue to supply, a healthy and necessary degree of stimulus and an influx of specialised techniques.

One of the biggest challenges facing our profession to-day is the supply of specialist workers for research and teaching and for an adequate laboratory diagnostic service. We are not keeping pace quantitatively with the general technological advance in the country, and many research projects of great fundamental and economic importance are not being tackled because of shortage of personnel. Here, once again, we have a field in which part of the solution lies in collaboration with medical scientists. The much greater size and width of experience of the medical profession has naturally led to the establishment of a more diverse and often more specialised laboratory organisation. The prospective gain to post-graduate training in this field is very large, and although it has been utilised to some extent in the past it could be developed considerably. The value of this course lies not only in the specialist training obtained, but also in the widening field of view and in the contacts made with medical scientists; this often flowers later as collaborative work, or as useful, stimulating and mutually advantageous consultation.

The financing of much veterinary research is done by the Agricultural Research Council. Some think it strange that, having made the decision to place our university Departments within the general medical orbit, we are dependent on the Agricultural Research Council and not the Medical Research Council for judgment of the suitability of research projects for monetary support. There are strong arguments on both sides, but despite the presence of several eminent medical men on the A.R.C. some people feel that it would be better to have research projects judged, and policy decisions made, by a body whose interests and abilities lie mainly in the broad field of medical science. If the question of whether we should have a separate Veterinary Research Council is not settled, serious consideration should be given to assessing which of the existing Councils would best serve the needs of veterinary research.

XUM

Studies on Anaemia in Pigs

IV—The Effect of Iron Administered either Intramuscularly or Orally, on the Live-weight of Suckling Pigs†

BY

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National Institute for Research in Dairying, Shinfield, Reading

SUMMARY.—There were no significant differences between the live-weights at 3 or at 8 weeks, of pigs given either a single intramuscular injection of an iron dextran preparation or an oral dose on 6 consecutive days of iron pyrophosphate, as prophylactic treatment against anaemia.

IN the first report of this series, Barber, Braude and Mitchell (1955) showed that for the prevention of anaemia in baby pigs, using blood haemoglobin levels as the criteria, the injection of an iron dextran preparation was as effective as oral dosing with iron pyrophosphate. Prophylactic treatment against anaemia using intramuscular injections of iron is now widely practised, and claims have been made that pigs given iron in this way are heavier at weaning than pigs given iron by mouth. To investigate this claim, an experiment was carried out to compare the growth rates of pigs given iron either intramuscularly or orally as preventive treatments for anaemia.

Experimental

The litters of 33 Large White sows having at least 6 pigs were weighed at 3 days of age, and, within each litter, pigs were paired according to weight. One member of each litter-mate pair received treatment A—a single intramuscular injection of 2 c.c. of an iron dextran preparation,* supplying 150 mg. iron, given on the 3rd day of life—while the other received treatment B—an oral dose of 2 c.c. iron pyrophosphate, supplying about 30 mg. iron, given daily for 6 consecutive days from the 3rd to the 8th day of life.

All the sows farrowed indoors in a Danish-type piggery and remained there for at least 1 week. Some litters stayed in the same house until weaning at 8 weeks, while others were transferred to huts with open runs permanently located on a concrete apron. The same routine of feeding and management applied to all the litters, and male pigs were castrated when 3 weeks old. From the 14th day of life all the pigs had access to a good quality creep feed.

The pigs were weighed at 21 and 56 days old.

Results

When one piglet died or failed to weigh more than 20 lb. at 56 days of age, the results for that piglet and its pair-mate were discarded. Thirty pairs from 15 litters were affected, 19 piglets (13 receiving treatment A and 6 receiving treatment B) died, and 15 piglets (9 on A and 6 on B) did not thrive. There was no indication that this casualty rate was influenced by the applied treatments.

The mean values for the 21- and 56-day live-weights of 114 piglet-pairs from 33 litters are given in Table I and the mean squares from the analyses of variance are shown in Table II. The small differences between mean values obtained for treatments A and B were not statistically significant at the 5 per cent. level. of probability.

TABLE I
MEAN VALUES FOR LIVEWEIGHT (lb. PER PIG)

Treatment	At 21 days of age	At 56 days of age
A (Iron dextran given by intramuscular injection)	12·07	35·63
B (Iron pyrophosphate given by mouth)	11·84	34·71
A—B	0·23±0·159	0·92±0·539
Significance of difference between treatment means	0·2 > P > 0·1	0·1 > P > 0·05

Standard errors with 81° of freedom.

TABLE II
MEAN SQUARES FROM THE ANALYSES OF VARIANCE

Item	Liveweight at 21 days		Liveweight at 56 days	
	d.f.	M.S.	d.f.	M.S.
Litters	32	8·35***	32	115·79***
Treatments ...	1	3·15	1	48·54
Litters × treatments	32	1·44	32	20·05
Blocks within litters	81	4·88***	81	35·59**
Remainder	81	1·44	81	16·57

** 0·01 > P > 0·001 ; *** 0·001 > P

As might be expected, highly significant differences in live-weight between litters and between blocks (*i.e.* litter-mate pairs) within litters were obtained.

Acknowledgment.—The authors wish to thank Messrs. Benger Laboratories Ltd. for the gift of Imposil.

Reference

BARBER, R. S., BRAUDE, R., & MITCHELL, K. G. (1955). *Vet. Rec.* **67**. 348.

* Imposil. Bengers Ltd.

†Part III of this paper was published in THE VETERINARY RECORD of January 4th, 1958 (*Vet. Rec.* **70**. 13).

Congress Paper

Experiences with a Scheme for Supervising Pig Herds Believed to be Free from Enzootic Pneumonia (Virus Pneumonia)*

BY

R. F. W. GOODWIN and P. WHITTLESTONE

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SUMMARY.—Earlier work in Britain on chronic respiratory disease in the pig is discussed, with particular reference to the approach to diagnosis and the development of an Association for the establishment of herds free from so-called virus pneumonia.

The veterinary surgeon's certificates, the farmer's declarations and the standards of the Association for the Advancement of Virus Pneumonia Free Pigs are given in the appendices. The reasoning behind the questions in the certificates and declarations, and also the requirements laid down by the standards, are explained.

Some of the respiratory-disease problems that have required differential diagnosis are described. One herd (SS) had a 16 per cent. incidence of small, consolidated lesions in the anterior lobes of the lungs from the fattening house. Histologically, the lesions in three-quarters of these affected lungs were indistinguishable from 3rd- or 4th-stage enzootic pneumonia. In 2 transmission experiments, only 3 out of 7 pigs inoculated via the respiratory route with lung suspension from a natural case had consolidated lesions; the latter were likewise very small macroscopically and even the pigs killed as early as 27 days after inoculation had lesions histologically indistinguishable from 4th-stage enzootic pneumonia. Hence, the character and low incidence of the lesions on the farm were paralleled in the experimental animals.

In a second herd (X1), an acute respiratory infection associated with extensive pneumonic lesions, and rhinitis, spread rapidly through many suckling litters. Clinically and histologically, this pneumonia showed differences from enzootic pneumonia. The disease in the young pigs ended abruptly and did not spread to older stock housed in the same buildings. Furthermore, when the affected suckling litters reached bacon weight, even though they had been fattened in large groups, they showed very contracted, healing lesions in the lungs that histologically, did not closely resemble any stage of experimentally-produced enzootic pneumonia. Repeated attempts to reproduce this pneumonia in young pigs were unsuccessful. The following year a clinically and histologically similar disease again swept through many litters of suckling pigs in the same herd. When the affected litters in this second outbreak reached bacon weight, however, they showed a high incidence of active-looking, consolidated lesions that closely resembled 2nd- to 3rd-stage enzootic pneumonia. Two other herds (L and D) established from herd X1 developed a severe disease in the suckling pigs that was clinically and histologically similar to the original disease in the latter herd. As in herd X1, all the adult stock in herds L and D remained clinically unaffected.

A third herd (MG) is mentioned where, in a period of 6 months, 46 per cent. of 395 lungs examined had consolidated lesions of the anterior lobes. These lesions were associated with a heavy lung-worm infection.

In each of 2 further herds (Q and DB) there was an individual pig that coughed persistently and at post-mortem *examination showed a pneumonia indistinguishable from experimentally-produced enzootic pneumonia. Both these pigs were closely housed with other pigs and, although a high percentage of the lungs from the in-contact animals were examined at slaughter, no comparable lesions were found.*

The last herd (TC) described appeared to have enzootic pneumonia in the fattening house, but a group of suckling litters affected with pneumonia were removed for study before they came into contact with the main part of the herd. The pneumonia in these animals showed consistent histological differences from both enzootic pneumonia and the pneumonia seen in the suckling pigs in herd X1; it was readily transmitted experimentally, when it largely retained its histological character and continued to differ from a strain of enzootic pneumonia transmitted in control animals.

The possible relationships between these various pneumonias seen in the field are discussed, and the implications of this work for the control of enzootic pneumonia and for wider health-control schemes in the pig are noted.

Introduction

RESPIRATORY diseases are common in pigs, and can become a problem under intensive conditions of rearing and fattening, when large numbers of animals are housed closely together. Within this respiratory disease complex, there is a chronic disease syndrome in the field, which is associated with a persisting pneumonia and in which long-standing and apparently recovered cases appear to remain infective to healthy pigs.

Pneumonic lungs collected from this general background of chronic respiratory disease have been used experimentally in Britain (Gulrajani & Beveridge, 1951a,b; Betts, 1953; Betts & Campbell, 1956; Pattison, 1956; Whittlestone, 1958; Whittlestone & Walzl, 1960), to produce pneumonias with lobular consolidations in the antero-inferior parts of the lung.

* Paper presented to the Annual Congress of the British Veterinary Association in Glasgow on Friday, September 2nd, 1960.

If these authors were all studying the same disease, it could be concluded that this type of pneumonia can be readily reproduced, but only via the respiratory route. The shortest incubation period, as judged by the development of pneumonia detectable by naked-eye examination, has been about 1 week; the lesions then gradually become more extensive. Histologically, there is a progressive development of an alveolar-cell pneumonia, mononuclear-cell accumulations associated with blood vessels and bronchioles, and, later, progressive lympho-reticular hyperplasia. Infected pigs have usually shown only mild clinical signs; recovery has been gradual, and in uncomplicated cases the disease has invariably been non-fatal. Lung suspensions have commonly produced pneumonia after being filtered through gradocol membranes of average pore diameter 0·8μ, and there is some evidence suggesting that the limiting pore diameter for producing macroscopic lesions with a similar histological picture is about 0·6μ. The causal agent also persists in the lungs for several weeks after infection and, with the antibiotics tried, establishment of pneumonia has been prevented only by the tetracyclines. Pneumonic tissue *in vitro* has been infective to pigs after 24 hours at 20° to 22° C., but not after 72 hours; it has similarly remained infective for 4 days at 4° C., but the limiting time for infectivity at this temperature has not been determined. On 2 occasions, an initially high concentration of infective lung suspension sprayed into premises that had also housed clinically affected pigs, did not infect susceptible pigs put in the same premises 24 hours later.

In Britain, Gulrajani & Beveridge (1951b) used the non-commital name "infectious pneumonia" for the disease that they had transmitted. This title was subsequently changed to "virus pneumonia" (Betts & Beveridge, 1952; Betts, 1952), a term that was also applied to pneumonias in the field. Comparable experimental and natural diseases abroad have been given several other names, one of which is "enzootic pneumonia". Because the gradocol-filtration and antibiotic-sensitivity experiments summarised above do not exclude a non-viral causative agent, we prefer this older name "enzootic pneumonia",* which is more descriptive but, once again, aetiologically neutral.

The exact relationship between the chronic respiratory diseases studied in different centres, however, is not known. Nor is it known to what extent the materials used for experimental transmission are representative of the problem as a whole in the field. Because of general similarities between the experimental and field diseases produced by material originating from different sources, however, there has been a tendency to think of all these chronic respiratory conditions as one disease, and among farmers, in particular, the term "virus pneumonia" often has a very loose meaning. As we cannot yet relate the different chronic respiratory infections of pigs described in different countries, we will confine our remarks in this paper entirely to the British literature and field conditions.

The experimental disease has been shown to affect the growth rate of pigs (Betts & Beveridge, 1953; Betts, Whittlestone, Beveridge, Taylor & Campbell, 1955), and it has become increasingly clear that herds free from chronic respiratory disease in the field can achieve higher levels of productive performance. So striking may be the difference between affected and unaffected herds under intensive husbandry conditions, that freedom from virus pneumonia became an essential requirement of a number of pig farmers in Britain. In due course, a small group of these farmers, who had been pioneers in the establishment of virus-pneumonia-free herds, formed an association termed the Association for the Advancement of Virus Pneumonia Free Pigs, with the object of spreading knowledge about virus pneumonia and describing minimum health standards for herds believed to be free of the disease. When the Association had been established for some time, the present authors were asked to advise it scientifically, to prepare its rules and standards, and to administer its veterinary affairs.

In considering this task, we were aware of several difficulties, both administrative and scientific. First, the Association was privately financed and could not afford to employ, in its early life at least, any full-time administrative staff. Secondly, it was primarily an Association of farmers, with the administration passing directly between an executive committee and the farming members. This meant that participation in the scheme by veterinary practitioners had to be on an indirect basis through the farmer, with the result that the veterinary profession has not received much information about the workings of the Association. A few veterinary surgeons have become associate members, in order to receive the Association's literature, but, in general, the aims and policies of the Association are not yet widely known. One of the main objects of this paper, therefore, is to summarise the present position for those veterinary surgeons who might be asked to supervise herds claiming freedom from virus pneumonia. The main difficulty confronting us, however, was that the Association was already committed to deal with a condition that had not been adequately defined and we were faced, therefore, with the scientific problem of working without any specific diagnostic methods. In spite of these various considerations, we agreed to undertake the veterinary responsibilities of the Association because, in the first place, we believed that the control scheme would have more chance of success if it were under close veterinary supervision, with the herd and lung examinations being made by veterinary surgeons, rather than by laymen. Secondly, being interested in the problem of respiratory disease in pigs, we saw the Association as a medium for providing both information and challenging problems in this field.

*As the Association for the Advancement of Virus Pneumonia Free Pigs embodies the term "virus pneumonia", it is simplest to use the same nomenclature in this paper when discussing the regulations and standards of the Association, but for more general discussion the name "enzootic pneumonia" will be used.

Detection of the Disease

The first decision to be made was whether only enzootic pneumonia should be controlled or whether the Association's regulations should embrace other respiratory diseases of pigs. There are obvious economic advantages in controlling other diseases, but even less is known about these and it was preferable, in the first instance, to include only enzootic pneumonia, where a certain amount of factual knowledge already existed. In addition, it is unlikely that regulations drafted to control enzootic pneumonia would be appropriate for another respiratory disease with, in all probability, a different pathology and epidemiology. Having reached this conclusion, the problem then was to devise regulations that would both detect and distinguish enzootic pneumonia.

Earlier workers in Britain identified the disease they were describing by the presence of macroscopic consolidation in the anterior lobes of the lungs. In this manner, it is probable that Blakemore & Gledhill (1941a, b) included at least two diseases in their description of swine influenza in East Anglia. Lamont (1952) has stated that in his description of swine influenza in Northern Ireland (Lamont, 1938), he likewise merged more than one disease. Unable to associate the swine influenza virus with pig pneumonias in East Anglia and elsewhere, Gulrajani & Beveridge (1951a, b) adopted the name "infectious pneumonia" for the condition that, on the basis of consolidated lesions in the anterior lobes of the lung, appeared to them to be widespread in the field. These lesions were taken to be typical by Betts (1952) and, by combining the incidence of such macroscopic lesions among fat pigs in slaughterhouses with the effects of the experimental disease produced by material from only one field source, Betts & Beveridge (1953) assessed the national economic loss from virus pneumonia. Thus from 1938, when Lamont stated that macroscopic lung lesions could be used to diagnose swine influenza in the field, until 1955, when consolidated lesions of the anterior lobes were still largely taken as evidence of virus pneumonia (Betts, Whittlestone & Beveridge, 1955; Barber, Braude, Mitchell & Betts, 1955; Whittlestone & Betts, 1955)—although histological differences between macroscopically similar lesions are mentioned—it is difficult to find in the British literature any appreciation of the concept that macroscopic lesions might result from a number of different processes. The presumption that the lesions of virus pneumonia could be identified macroscopically was strongly questioned by Pattison (1956), who pointed out that consolidated lesions in the anterior lobes of the lungs of pigs, showed such a variety of histological pictures that it appeared certain that other aetiological agents were involved. He also recognised that the lymphoid hyperplastic reaction produced in the so-called virus pneumonia might occur in other conditions. These views of Pattison were supported by Whittlestone (1957, 1958), and our joint field studies before drafting the Association's rules had progressively strengthened us in the view that enzootic pneumonia could not be diagnosed on pathological features alone.

Faced by this less tidy approach to respiratory disease in the pig, and prompted by the perplexing diseases being encountered in the field (which are described later), we have sought new base-line definitions for the entity that we are attempting to control. These definitions are for use at the consultant level and are a little different in conception from those in current routine use in the veterinary surgeon's certificate, which are described later when explaining the regulations. The first criterion chosen for re-appraisal was the histological picture, as this was already incorporated as a diagnostic tool in the Association. To make this histological assessment, it was necessary to define enzootic pneumonia in some other, non-pathological, way and it seemed that a clinical differentiation was the best starting point available; for a number of pneumonias sharing major histological features appeared to one of us to differ clinically and epidemiologically. We have always borne in mind, however, the danger arising from the fact that enzootic pneumonia might manifest itself in the field in widely different forms. Nevertheless, a start had to be made somewhere, and the following general conditions were laid down for the provision of pneumonic material for histological examination.

First, the herd should have been under our supervision and free of pneumonias for an adequate period. Secondly, the herd should become naturally infected with a type of pneumonia that clinically simulated the disease studied experimentally as virus pneumonia. In particular, the adult animals should be seriously affected, as this might indicate a population that was initially uninfluenced by specific immunity. Thirdly, after the acute phase had subsided, the growing pigs should suffer from a chronic pneumonia that, if studied experimentally, would have the same gradocol-filtration and antibiotic-sensitivity characteristics, etc., as experimentally-produced virus pneumonia. Not surprisingly, these exacting requirements for the procurement of uncomplicated field cases of enzootic pneumonia have not yet been fulfilled, but some have been obtained from 5 outbreaks that have partly fulfilled the above criteria. A range of cases, both acute and chronic, is being sought, in order to amplify the histological knowledge that has been gained experimentally (which forms the basis of the histological classification in the veterinary surgeon's certificate), because for field diagnosis it is preferable to know all the manifestations of a disease. To date, the examination of this specially selected field material has given no cause for a change of view on the progressive histology of enzootic pneumonia.

The Present Regulations

These regulations are given in Appendix 1, A and B, and the reasoning behind them can best be illustrated by examining the requirements stage by stage. Although some herds had been claiming freedom from virus pneumonia for several years, the regulations first came into operation on January 1st, 1959, and no herd can claim time before this date to be recognised for compliance with the Association's rules. The veterinary surgeon's certificate

and the farmer's declaration are signed at the end of each 6-monthly period and forwarded to the Association. Each of these periods is numbered consecutively and, hence, we are now in period 4 of the scheme.

Because we believe that virus pneumonia is much more difficult to detect in herds without an intensive system of husbandry (that is, herds without a fattening house) there are separate regulations (Appendix 1, B) for these herds and they are accorded a lower status in the Association. It will be seen from the standards (Appendix 2) that herds complying under the non-fattening-house regulations are listed separately and any farmer buying breeding stock from such a herd must accept the transfer of the vendor's lower status.

The regulations and standards printed in the appendices are those applying to period 3 (January to July, 1960). All these forms went through several editions before 1959 and, as a result of experience in periods 1 and 2, they were again extensively modified for period 3. Most of the modifications, however, were made either to clarify ambiguities of wording or to seek more detailed information about the respiratory disease status in a herd. As yet, little alteration has been necessary to the basic framework of the regulations.

All the regulations described below were drawn up on the assumption that virus pneumonia, while not necessarily producing pneumonia in every pig in a herd, was primarily manifested as a pneumonic condition. If the causal agent can exist in a herd without producing pneumonic lesions, these regulations could not detect it. There is no evidence that the latter state occurs, but the establishment of many virus-pneumonia-free herds may help to test this assumption. There are other basic assumptions inherent in the regulations that will likewise be tested by experience, so that certain sections of the rules are necessarily exploratory, and they will require modification if they can no longer be supported against accumulating scientific evidence.

Regulations for Herds with a Fattening House (Appendix 1, A)

Veterinary Surgeon's Certificate

Question 1. The object here is to test every breeding female for the ability to transmit infection to her litter. To help those farmers who cannot accommodate their whole output of weaners in a fattening house, the regulations state that only one-third of each litter need be passed into a fattening house. This figure of one-third is one of several arbitrary decisions that had to be made in the light of our past experience; it may have to be modified. The weaners must pass into the fattening house before 3 months of age, so that there is little chance of them becoming non-infective before they are put in contact with other pigs. All pigs entering the fattening house must remain there for 2 months, so that there is a good chance of them infecting other animals. A fattening house is defined.

Question 2. Having sampled the litter of every dam and mixed the samples in close confinement, the output from the fattening house is sampled for the presence of pneumonia. We are not concerned that the output sample be related (except by contact) to the input sample, as these 2 samples are serving separate purposes. Ideally, lung examinations should be made continuously throughout each year, but for practical reasons, these particular regulations operate only on a quarterly basis. In each quarter the lungs from either (a) half the output, or (b) 30 pigs, must be examined *post mortem* by a veterinary surgeon. The examination of lungs by any other person, such as a non-veterinary meat inspector, is not acceptable. The term output is defined. When the output from the fattening house is small, a relatively large sample is taken, but once the quarterly output exceeds 60 pigs, the number of lung examinations remains fixed at 30. We feel that special factors begin to operate in very large fattening houses, so that the sample of lungs from 30 pigs is then adequate.

Questions 3 and 4. These are self-explanatory. It proved necessary to define a lung.

Question 5. The veterinary surgeon examining the lungs is asked to record the incidence of consolidated lesions in the anterior lobes, the extent of which is defined. He is not required to differentiate these lesions macroscopically, or to judge whether they might be caused by virus pneumonia. Only the anterior lobes are assessed, because virus pneumonia rarely produces lesions outside this prescribed area without causing lesions within the area at the same time. By excluding the major portions of the diaphragmatic lobes, other lesions—notably those associated with lung-worms—are commonly avoided. If there are consolidated lesions in the anterior lobes, one sample from each lesion, up to a maximum of 4 blocks per lung, is retained in formol saline in case histological examination is necessary later (see Question 7). The veterinary surgeon making the lung examinations need not be the same as the one answering the other questions on the certificate. Only one veterinary surgeon must sign the certificate, however, but he can use information provided by a second veterinary surgeon, as when the lungs are examined in a distant bacon factory.

Question 6. In most pig herds, occasional pneumonias will occur and it did not seem practicable to require that all the lungs examined must be free of consolidated lesions of the anterior lobes. Under intensive husbandry conditions, however, a herd chronically affected with enzootic pneumonia will, in our experience, usually have more than 20 per cent. (and often about 40 to 50 per cent.) of the lungs affected at slaughter. In severely affected herds this figure can increase to nearly 100 per cent. The figure of 5 per cent. was chosen, therefore, as a cautious safety limit. If the incidence of consolidated lesions is less than 5 per cent., the herd passes this part of the regulations in the period concerned. We cannot say definitely at the moment, however, whether it is possible for virus pneumonia to persist in a fattening house for 18 months or more without causing more than 5 per cent. of consolidated lesions of the anterior lobes.

Question 7. If the incidence of consolidated lesions of the anterior lobes exceeds 5 per cent., the first safety level has been exceeded and all the lesions must be examined histologically. As there must be uniformity of interpretation, a laboratory had to be found that would undertake the histological work. Fortunately, the Livestock Research Centre of the Animal Health Trust, Stock, Essex, offered to provide this service and their histological classification conforms to a prescribed pattern. For simplicity, histological reports from other laboratories are not accepted at this stage of the Association's development. Any widening of laboratory facilities in the future would still require a standardised method of histological assessment.

When the Association started, it was known that experimental cases of enzootic pneumonia produced with material originating from several British sources showed a similar histological picture, and that this picture was also commonly seen in chronic pneumonias in the field (Whittlestone, 1958). The histological classification used at the outset, therefore, was based on a combined assessment of these field and experimental findings. As has been explained earlier in this paper, the histology is studied in more detail at the consultant level and the interpretation is then much more flexible than the relatively rigid classification necessary for preliminary screening. Any modification of approach resulting from further work would require time to be substantiated to the point where it could be incorporated into the routine side of the histological reporting, so there are always 2 types of histological classification in concurrent use in the Association, and the reports made by the Animal Health Trust mainly serve to indicate when the pneumonias in a herd require further investigation. The routine categories are as follows: —

I. INDISTINGUISHABLE FROM VIRUS PNEUMONIA.

In this category are the changes seen at the 3 main stages in the development of the macroscopic lesion:

(a) an alveolar-cell pneumonia with mononuclear-cell accumulations in the interstitial tissues, particularly in the perivascular, and sometimes in the peribronchiolar and subpleural areas,

(b) a similar, but less marked alveolar-cell pneumonia with more marked and dense mononuclear-cell accumulations, but particularly with peribronchiolar lympho-reticular hyperplasia, and

(c) few or no areas of alveolar-cell pneumonia, but marked lympho-reticular hyperplasia, particularly affecting the bronchial tree.

Any intermediate stage between these 3 subdivisions is likewise classed as indistinguishable. (Stages (a), (b) and (c) above are the same as the 2nd 3rd and 4th stages, respectively, of the J strain of enzootic pneumonia described by Whittlestone & Walzl, 1960.)

II. SUSPICIOUS OF VIRUS PNEUMONIA.

In this category are included any pneumonias that share any of the features described in I, but which do not closely resemble the sub-divisions (a), (b) or (c), or any of the progressive stages between them.

III. NOT SUSPICIOUS OF VIRUS PNEUMONIA.

In this category are those consolidated lesions that show none of the prime features described in category I.

Where the line is drawn between these categories, and particularly between categories II and III, is to some extent a personal decision, and hence the necessity for prior agreement in the matter.

Question 8. Because the histologolical reports must inevitably lag behind the lung examinations, provision is made in this question for completing records outstanding from the previous period.

Questions 9, 10 and 11. These are self-explanatory.

Farmer's Declaration

Questions 1 and 2. These questions supply details of pig movements that can be co-ordinated centrally. They act as a check on the degree to which herds have had contact with other herds and they provide a complete record of movements within the Association for observing the spread of any respiratory conditions, such as those that might be confusing the situation.

Questions 3 and 4. These allow the farmer to record any possibility of an inadvertent contact with possibly infected stock. A contact is defined, both as regards distance and time. The time interval of 14 days for premises or transport that is not disinfected, may seem excessive; but we have in mind here winter conditions, and the causal agent is known to be infective after at least 4 days at 4° C.

Questions 5, 7 and 8. These are self-explanatory.

Question 6. To assist the detection of virus pneumonia, the conditions of husbandry should be as intensive as possible. The degree of intensification depends not only on the concentration of pigs in a confined space, but also on the continuity of throughput. By ensuring that the fattening house is never cleared of pigs, every encouragement is given to the build-up of the disease, should it be present.

Regulations for Herds without a Fattening House (Appendix 1, B)

Veterinary Surgeon's Certificate

Question 1. The problem in herds of this type is to assess the respiratory disease status of the breeding stock from litters reared in isolated groups. Every group of pigs, therefore, must be examined as a unit and the reliability of the herd assessment is only as strong as the weakest of the individual group assessments. In period 3, it was necessary to examine the lungs of at least 30 per cent. of the animals in a group, when 3 or more litters were mixed together; or of 50 per cent. of the group, when only one or two litters formed the group. From the revised standards of the Association (Appendix 2), however, it will be seen that these regulations have been stiffened. The earlier figures of 30 and 50 per cent. have been increased to 50 and 66 per cent. respectively, in period 4, as we had evidence

that it might be possible for over half the animals in an isolated group to have resolved their pneumonic lesions completely by the time of slaughter. There is a slight let-out clause in these severe requirements, in that where it would be inconvenient to slaughter as many as 66 per cent. of the animals in a litter, the percentage may be reduced to 50 per cent. again, providing the animals are slaughtered between 14 and 18 weeks of age.

Question 2. With this less intensive system of husbandry, we feel that every consolidated lesion of the anterior lobes indicates a potential danger and hence all these lesions must be sent for microscopic examination.

Questions 3, 4 and 5 are self-explanatory.

Farmer's Declaration

This declaration is essentially the same as the one for herds with a fattening house, except that certain questions that are no longer applicable are omitted.

The Standards of the Association

These standards describe the requirements that must be fulfilled for a herd to claim freedom from virus pneumonia under the Rules of the Association. Under paragraph 1 (a), it will be seen that a herd can claim freedom by carrying out the necessary lung examinations, etc., for 3 consecutive periods; that is, for a period of 18 months. The reason for this long preliminary testing period is to try ensure that any intermittent carriers of the causal organism among the adult population have sufficient time to demonstrate their presence. It is not known, however, whether apparently recovered adults can become infective later in life. When a herd has been established entirely from other herds that have already satisfied the Association's requirements—paragraph 1 (b)—this testing time of 18 months is shortened, providing that the new herd has been established without breaking the contact rules.

Paragraph 2 (iii) (b) explains the procedure when, in herds with a fattening house, the first safety limit of 5 per cent. of consolidated lesions is exceeded. All the consolidated lesions must then be examined histologically and, as explained earlier, veterinary surgeons carrying out lung examinations (either for their own clients, or at a bacon factory on behalf of other veterinary surgeons) must retain their samples, fixed in formol saline, until the end of each period, when it will be known whether microscopic examination is called for or not. If 75 per cent. or more of the lungs examined histologically are officially classified as not suspicious, then, in the absence of any positive evidence for the presence of virus pneumonia, the herd is automatically cleared. If this cannot be done, the herd becomes a special case that must be investigated in detail by an approved consultant of the Association.

Paragraph 2, second section, (ii), states that, in herds without a fattening house, no hard and fast rule can yet be made for passing herds with pneumonias of the anterior lobes. Sometimes they can be cleared on histological examinations alone, but often they require a consultant opinion.

Paragraph 4 allows for special provisions where farmers wish to introduce new blood from herds outside the Association. It has been agreed that the Association must, wherever possible, legislate for progress and make special efforts not to hinder the introduction of blood lines possessing other good qualities. Where the proposed manoeuvre carries a slight risk, it must be officially supervised by the Association as here explained, and until such time as the manoeuvre has been shown to have been completed to the satisfaction of the Association, the herd is placed on the supervised list (List B). Herds cannot be included in List B unless they have complied with all the other regulations of the Association and show no evidence of infection with virus pneumonia.

Paragraph 5 permits the publication of 2 herd lists by the Association. The first, List A, shows those herds that have complied with all the Association's standards, whereas List B records the herds under supervision. List B is a lower status than List A and hence herds in List A that receive pigs from herds in List B collect the latter status.

A footnote to the standards stresses that compliance with the standards of the Association carries no guarantee of freedom from virus pneumonia. In the first instance, there must always be a time lag between the assessment of a herd and its current status. During this time, the herd might be incubating the disease. Secondly, any disease-control scheme such as this can only be as reliable as the integrity and conscientiousness of the veterinary surgeon and farmer signing the certificates and declarations. It might be pointed out here that the proper signing of the certificates of the Association might well require several hours' preliminary work on the farm. Upon the accurate signing of these documents a great deal may depend, as the sale of animals from an infected herd can soon ruin years of work in the herds to which the infected animals are sold. Thirdly, the regulations themselves might not yet cater for all the natural variations of the disease.

Differential Diagnosis

In the last section, it was pointed out that the regulations cannot cater for all situations and there is a deliberately chosen point beyond which special investigations have to be undertaken to assess the status of a herd. This re-assessment of the situation is made at the moment by one of us (R.F.W.G.), who inspects the herd and collects the relevant data. Use may be made of the practitioner's reports at this stage, but it is often necessary to examine further batches of lungs *de novo*. The result of the whole investigation is then passed on to the executive committee of the Association for ratification. This ratification is important, especially as it is sometimes necessary to make a political decision on what degree of risk the Association should take in doubtful cases. Although the consultant verdict is reached singly, it is made against the background, and always within the limits, of the authors' joint views on the disease. In coming to a decision on any particular herd, all

the laboratory and clinical findings, together with any results that may be available of experimental transmissions, are considered together. As it is often useful to have an early clinical assessment of an outbreak of respiratory disease that may later require differentiation from enzootic pneumonia, and as it is important to recognise as soon as possible the existence of enzootic pneumonia in a herd selling breeding stock, it is suggested that if a problem of this type arises in what should be a virus-pneumonia-free herd, advice should be sought through the Association without delay. The difficulties of differential diagnosis might have been expected to reach us mainly by this consultant channel, but, so far, this has not been the main source of our diagnostic problems; these have arisen in the following way.

Several practitioners have found it inconvenient to undertake the supervision of herds for the Association. In some instances the reason has been the lack of a suitable bacon factory; in others, it has been clear that a great deal was at stake in the establishment of the herd and the subsequent sale of breeding stock, and they preferred to transfer their responsibility elsewhere. For these and other reasons, one of us (R.F.W.G.) has taken over the responsibility of a number of herds widely spaced in the country, and has carried out in these herds, on behalf of practitioners, all the clinical inspections and lung examinations required by the Association. This work has not only kept us closely in touch with the difficulties of making our own regulations work on the farm, but it has provided us with nearly all our problems in differential diagnosis. Some of these will now be discussed, herd by herd, and in presenting this material it should be emphasised that it has been in our hands from the beginning; for, not only was the field side already under our own control, but all the histological investigations have likewise been made by one of us (P.W.).

Herd SS

This herd was first visited in May, 1959, because the owner believed his herd to be free from enzootic pneumonia, even though he had taken no positive steps to this end, and he wanted to be satisfied that this was so. Adult pigs that had entered the herd in the past from another herd, known to be enzootic-pneumonia-free, had not contracted any obvious respiratory disease, and at this visit and 2 other later visits virtually no sign of respiratory disease could be elicited in either the farrowing or the fattening house. The fattening house had held pigs continuously for about 2 years.

Lung examinations commenced in May and continued until August, by which time the lungs of 75 routinely-slaughtered bacon pigs had been inspected. Of these, 12 coming from different litters showed consolidated lesions in the anterior lobes—an incidence of 16 per cent. Two-thirds of the lesions were small (less than 2 cm. across) and the remainder did not exceed 4 cm. Although rather limited in extent, they were macroscopically similar to the lesions seen in enzootic pneumonia; all 12 were examined histologically.

Seven showed moderate to very marked lympho-reticular hyperplasia, often with partial obliteration of the bronchiolar lumina. In all but one of these, alveolar-cell proliferation and desquamation was very slight, but all showed varying degrees of mononuclear-cell accumulations around the blood vessels and bronchioles. Thus this group (Fig. 2) were indistinguishable from the 4th stage of enzootic pneumonia.

In the eighth animal (1488), there was a single lesion (occupying about one quarter of the intermediate lobe) in which all areas showed an alveolar-cell pneumonia, marked mononuclear-cell accumulations, particularly in the perivascular tissues and the lamina propria of the bronchioles; this, together with marked lympho-reticular hyperplasia, caused some obliteration of the bronchiolar lumina. The whole lesion, therefore, closely resembled the 3rd stage of enzootic pneumonia, and it was used for the experimental transmissions described later. The ninth pig showed similar but less marked changes.

The remaining 3 lungs showed slight to moderate patchy proliferation of the alveolar cells, but few free alveolar cells; there were small accumulations of mononuclear cells around some blood vessels and bronchioles. Thus, while these 3 shared some features in common with enzootic pneumonia they did not closely resemble any stage in experimentally-produced enzootic pneumonia.

Experimental transmissions. In the first transmission experiment, 4 porker pigs, probably free of enzootic pneumonia, were inoculated intratracheally with 20 ml. of lung suspension in broth (2 with a 10^{-2} dilution, and 2 with a 10^{-4} dilution). All the animals were killed 27 days later. One pig in each group had extensive pleurisy, but neither of these showed consolidated lesions in the lungs. The other 2 pigs had abscesses in the lung: one had chronic pleurisy, and the other, which had received the more concentrated inoculum, showed several small consolidated lesions in the anterior lobes. Histologically, these latter lesions showed both marked peribronchiolar lympho-reticular hyperplasia and mononuclear-cell accumulations in the lamina propria, which caused obliteration of some of the bronchiolar lumina. Some areas showed perivascular mononuclear-cell accumulations, and in a few areas there was a moderate increase in the number of alveolar cells, some of which were free in the alveolar lumina. Thus the lesions in this latter pig were histologically indistinguishable from a 4th-stage enzootic pneumonia.

The same lung tissue (1488) was used in the second experiment, when 3 MH pigs* ($4\frac{1}{2}$ months old) were each inoculated with 17 ml. of a 1 in 120 dilution intratracheally and 3 ml. of a 1 in 25 dilution intra-

*Two main types of pigs will be referred to in our experimental work. The first are those produced following hysterectomy according to the description of Betts, Lamont & Littlewort (1960). They are here referred to as CD pigs (colostrum deprived). The others come from a herd that was established from CD pigs and kept on the School farm. These animals are often referred to as "minimal disease" pigs, but this is a limitless term and hence we have designated them MH here, these being merely the initial letters of the farm.

nasally. No macroscopic abnormalities were detected in 1 pig killed 20 days later but, histologically, there were perivascular and peribronchiolar accumulations of mononuclear cells. The other pigs, killed 27 and 50 days after inoculation, both had minute consolidated lesions in the anterior lobes. Histologically, these showed moderate lympho-reticular hyperplasia and mononuclear-cell accumulations in the lamina propria of the bronchioles, obliterating the lumina in some areas. Apart from focal partial collapse, the alveoli were mainly normal. Thus, once again, the experimental pigs had lesions that were histologically indistinguishable from a 4th-stage enzootic pneumonia.

Pleomorphic organisms (P.O.)* could not be found in touch preparations made from the 5 field cases examined in this respect, nor in the 3 experimental cases with consolidated lesions of the anterior lobes.

Hence in these experiments, the pigs inoculated with material from herd SS showed, at slaughter, in addition to abscesses and pleurisy, a low incidence (3 out of 7) of small consolidated lesions, which macroscopically and microscopically closely resembled the most common lesion (4th-stage enzootic pneumonia) seen—also in low incidence—in the lungs from the farm. The strains of enzootic pneumonia studied experimentally to date, however, have differed from the type SS pneumonia, in that they have been more readily reproducible (besides resulting in more extensive lesions) and that 4th-stage lesions have not been seen as early as 27 days after inoculation.

Herd XI

This herd was mainly established during July to September, 1958, from stock believed to be free of enzootic pneumonia. The first litter was born in September, and others followed in close succession. Early in December, acute respiratory disease appeared in a litter 6 weeks old in a large farrowing house, and the condition spread rapidly to 13 other litters in the same house, as well as to 2 litters in a second farrowing house. Sneezing and loss of bloom were the first signs, followed by laboured abdominal breathing and a fairly characteristic throaty cough, which often sounded as if the piglet were about to vomit. Most litters were first clinically affected by 2 to 3 weeks of age, none were affected below 2 weeks of age and, in all, 16 litters became clinically ill. The standard of pigmanship in this herd, however, was abnormally high and in most cases the animals were beginning to recover about 1 week after first sneezing. Relatively few pigs remained unthrifty beyond weaning age.

The last litter to be affected was born in mid-December, and all subsequent litters were clinically normal. This apparent sudden limitation to the spread of infection was striking. Thus a total of 8 litters born during the 3 weeks after the birth of the last affected litter, and reared in the same small, enclosed farrowing house, appeared to remain free of the disease. Furthermore, when the recovered litters passed into a large fattening house as weaners, no respiratory disease appeared later in the older pigs from unaffected litters already there. At no time did any adult animals, such as the dams of the affected litters, show any sign of respiratory disease or more generalised ill-health. When the farm was visited at the end of February, there was no evidence of respiratory disease in two large fattening houses, in some subsidiary fattening accommodation, in the farrowing houses, or in any groups of adults; and only a few stunted pigs in one box indicated that there had been any previous trouble.

Nine pigs that were removed from the farm during the outbreak were later examined *post mortem*, at varying ages from nearly 4 weeks to 10½ months. Seven of them were killed or died before the age of 12 weeks and 5 of the snouts showed varying degrees of catarrhal to purulent rhinitis, with marked hypoplasia of the turbinates in 3. Six of these 7 animals had active pneumonic lesions, which were extensive in 4 (all the lobes being affected).

Histologically, in all the pigs there were small to moderate accumulations of mononuclear cells in the lamina propria of the nasal mucosa, often associated with the glands. No intranuclear inclusions similar to those described by Done (1955) were detected. Hereafter in this paper, bodies of this type will be referred to as I.N.I. (intra-nuclear inclusions). The pneumonias in the 6 pigs under 12 weeks of age were all histologically similar. In the 2 oldest animals (8 and 11½ weeks), there were areas in which the alveolar walls were thickened by many layers of alveolar cells, but all 6 showed an alveolar-cell pneumonia. Many alveoli and bronchioles contained a purulent exudate and there was epithelial hyperplasia of most bronchioles. In all areas there were moderate perivascular accumulations of mononuclear cells and, in some areas, very slight peribronchiolar mononuclear-cell accumulations and lympho-reticular hyperplasia. Thus, while these pneumonias shared many features in common with enzootic pneumonia, they differed from enzootic pneumonia, in that there was much more thickening of the alveolar walls with proliferated alveolar cells, and there was more hyperplasia of the bronchiolar epithelium (Figs. 3 and 4).

Satisfactory touch preparations were obtained from 6 of the 7 pigs under 12 weeks of age. In none of these were P.O. found. In 3 of the lungs, however, there were many organisms that differed morphologically from P.O., only in that they were regularly solid, coccobacillary and deeper-staining.

The examination of lungs from bacon pigs started in mid-March and by mid-November 157 had been inspected. Only 13 had consolidated lesions of the anterior lobes. The first 10 of these latter lungs

* The term "pleomorphic organism" is used to denote the structures seen in touch preparation of experimentally-produced enzootic pneumonia (Whittlestone, 1958). The name refers only to elements of this morphological appearance and there is no implication that they are a single entity. In summary, these delicate structures in air-dried, methyl-alcohol-fixed, Giemsa-stained preparations appear as pale, reddish-purple rings, triangles, bipolar forms, cocci and rods, usually varying in size from about 0·2μ to 1·0μ. Apart from those occurring in the cytoplasm of neutrophil polymorphs, they are mainly extracellular. In this paper, pleomorphic organisms of the above morphological appearance will be subsequently referred to as "P.O.".

were received between April and June; they all came from pigs that had been in affected litters, and they all showed the same macroscopic picture. The lesions were severely contracted, with virtually no obvious alveolar consolidation; the contractions covered large areas and formed such deep fissures that they split the anterior lobes into numerous, almost isolated, portions—connected only by thin sheets of tissue. Two piglets removed from the farm during the outbreak and kept in isolation until slaughtered at 10½ months of age, showed lesions identical with those occurring in this particular group of baconers from the farm.

Histologically, the above 12 lungs with contracted lesions showed partial alveolar collapse, thickening of alveolar walls (mainly with connective-tissue elements), and marked fibrosis of the interlobular septa, as well as the peribronchiolar and perivascular tissues. The alveoli contained very few alveolar cells and in most lungs there were areas with very small accumulations of mononuclear cells around the blood vessels and bronchioles. In one lung these accumulations were more marked and there was alveolar-cell enlargement, while in another there were small areas of marked lympho-reticular hyperplasia causing obliteration of the bronchiolar lumina. The latter lung, therefore, was histologically indistinguishable from a 4th-stage enzootic pneumonia but the other 11, while sharing some features in common with enzootic pneumonia, did not resemble any stage of the experimentally-produced disease.

Of the 70 lungs examined between September and November, only 3 showed consolidated lesions. The first 2 (examined in September) had very small consolidated areas which, microscopically, showed lympho-reticular hyperplasia and perivascular mononuclear-cell accumulations, but to a markedly different degree in each; so that one was histologically indistinguishable from a 4th-stage enzootic pneumonia, while the other was a similar lesion with less severe changes. The third lung (received in late September) had multiple, small, diffuse lesions which, on histological examination, showed heavy infiltrations with eosinophils (and also some mononuclear cells) in the bronchiolar walls, the perivascular and peribronchiolar areas, the interlobular septa and the alveoli; so it did not resemble enzootic pneumonia.

Hence, the clinical disease in the suckling pigs had departed almost as dramatically as it had arisen, and when the pigs from affected litters reached bacon weight they showed, at low incidence, a consistent macroscopic picture of an almost healed pneumonia. At this point, therefore, the disease appeared clinically different from enzootic pneumonia and this difference was underlined by the later examination of lungs from the fattening pigs: 157 lungs had been inspected during a period of 8 months and only 3 of these showed lesions similar to enzootic pneumonia. These latter lesions were comparable with a 4th-stage enzootic pneumonia (one of them being mild) but the affected areas were very limited.

The respiratory-disease situation continued to remain clinically quiescent throughout the summer farrowings of the sows that had had affected litters in the previous winter, although the owner believed that a few of these litters showed significantly uneven weaning weights when compared with the high degree of uniformity to which he was accustomed. The farm was visited again at the end of June and virtually no sign of respiratory disease could be found among the large number of pigs on the premises. Early in December, 1959, however, a disease that was clinically identical with that seen in the previous winter appeared in the litters in the main farrowing house, but the older stock remained clinically normal as before. By January, about 12 litters were ill and half of these were from sows whose litters appeared to have escaped the disease before. This is probably not very significant, however, because the pattern of spread in the farrowing houses during both outbreaks suggested that once the disease occurred it spread among the litters, rather than from each sow to its own litter. By February, about 15 litters had been affected but, thereafter, the disease once again disappeared. In April, about 150 pigs in one fattening house looked very healthy, and about 220 in a second house looked equally well. In the latter building, however, were the fattening pigs that had been affected when on the sow, and 3 of these fatteners still suffered from a loose, throaty cough.

In January, 1960, 7 pigs in various stages of the disease were removed from the farm. At autopsy, they varied in age from 1 to 8 weeks, but the pathology is best correlated with the time since the development of clinical signs, as this is known fairly accurately. One animal was selected because it was judged to be incubating the disease, even though it was not yet sneezing or coughing; it had a slight rhinitis but no macroscopic lesions in the lungs when killed at 7 days of age. On histological examination, however, the changes in the alveoli fitted into the more advanced general pattern of the others, but were small and patchy. The remaining 6 animals had been clinically affected for times varying between a few days and about 5 weeks; the one briefly affected showed a few small areas of pneumonia, but the remainder had extensive pneumonic lesions affecting all the lobes. Histologically, these pneumonias showed graded changes depending on the time that the animals had been clinically ill, and the over-all picture was almost identical with that seen in the young pigs in the first outbreak.

Satisfactory touch preparations were obtained from 6 of these pigs and many P.O. were found in 2 of them. This was the first detection of P.O. in the respiratory disease problems described in this herd.

The turbinates of these pigs likewise showed graded changes, varying from a slight catarrhal rhinitis with a suspicion of hypoplasia to a purulent rhinitis with marked hypoplasia; obvious hypoplasia was present in 5 of the 6 pigs that were clearly affected clinically. Histologically, all the animals showed small to moderate mononuclear-cell accumulations in the lamina propria of the nasal mucosa, together with intra-epithelial ab-

scesses. I.N.I. were found in 7 of 9 blocks examined from 1 piglet (an early clinical case when killed).

The examination of lungs from bacon pigs recommenced in January, 1960, when 1 out of 25 showed an extensive contracted pneumonia that was rather different macroscopically from the very contracted pneumonias seen between April and June, 1959. Histologically, it showed marked alveolar collapse and small areas of alveolar-cell pneumonia. There were moderate to marked mononuclear-cell accumulations and lympho-reticular hyperplasia peribronchially (causing partial obliteration of the bronchiolar lumina) but hardly any mononuclear-cell accumulations round the blood vessels. In April, however, 7 out of 22 lungs had pneumonia, and in May, 16 out of 21 were affected. The pneumonias in these last 23 cases were very similar to each other macroscopically and, in a sense, opposite from those seen at the same stage in the previous outbreak. Thus they appeared very active and oedematous, with ill-defined limits to the consolidated areas. So far, only the April cases have been examined histologically. One lung, and areas of 2 others, showed accumulations of eosinophils and mononuclear cells in the interstitial tissues. Of the other 6, 5 showed an alveolar-cell pneumonia, with small numbers of multi-nucleated cells in the alveolar lumina, but all showed moderate to marked perivascular and peribronchiolar mononuclear - cell accumulations, lympho-reticular hyperplasia (which in one instance caused obliteration of the bronchiolar lumina), and patchy catarrhal bronchitis. The latter 6 lungs, therefore, showed uniform changes and, not only were the lesions now very large, but they were indistinguishable from 2nd- to 3rd-stage enzootic pneumonia. There were, however, more multinucleated cells in the alveoli than is usual in experimental cases of the latter disease. P.O. were detected in all the 7 pneumonias received in April.

Hence, in contrast to the findings in the bacon pigs during the previous year, the lesions in this group of bacon pigs were both macroscopically and microscopically indistinguishable from enzootic pneumonia.

Experimental transmissions. It has proved difficult to transmit experimentally the pneumonia occurring in the suckling pigs in this herd. Field cases from the first outbreak did not produce macroscopic lung lesions in healthy pigs housed in the same cubicle. Neither was pneumonia produced by intra-tracheal inoculations of a lung suspension, by spraying CD pigs with a lung suspension, or by spraying CD and MH pigs with mixed suspensions of lung and turbinate material from 3 field cases. No I.N.I. were detected in these experiments.

Limited experimental transmissions with material taken from suckling pigs in the second outbreak, however, were more successful. Three MH pigs, 5 days old, housed in contact with 3 natural cases, were examined *post mortem* 7, 16 and 27 days later. Only the pig killed last had macroscopic lung lesions: there was an extensive pneumonia affecting all lobes which, histologically, was indistinguishable from a 2nd-stage enzootic pneumonia with secondary bacterial infection but, in addition, there were some multinucleated cells in the alveoli and patchy thickening of the alveolar walls with alveolar cells. P.O. and bacteria were found in touch preparations of the lung, but I.N.I. were not detected in the nasal mucosa of this animal, nor in the other 2 killed earlier.

Herds L and D

These 2 herds are mentioned because they were established largely from herd XI, and in both of them an acute respiratory disease that was clinically similar to that in herd XI appeared in the suckling pigs.

In herd D, the first farrowing was in July, 1959, but the disease did not appear until November—by which time the first farrowing sequence was ending and only a few litters were badly affected. Coughing spread widely, however, and unlike herd XI extended to the fattening house where, in January, 1960, out of a total of 82 animals, there were pigs coughing in every pen. One young coughing pig was killed and it showed a moderately extensive pneumonia which, histologically, closely resembled the changes seen in the young pigs in herd XI. No P.O. could be seen in these lesions.

Herd L was established between May and September, 1959. Farrowing started in June, and by September the weaning weights were progressively falling. Sneezing and coughing started in a litter in early October and then continued in every one of the next 14 litters. The disease became progressively worse in the later litters and persisted in the fattening house to such an extent that the herd became uneconomic and was completely liquidated in the summer of 1960. Pathologically, the lesions in 10 young pigs (the lungs of 6 being examined histologically) again paralleled the changes seen in the young pigs in herd XI. P.O. have not been found in any of the 5 lungs so far examined.

In herd L and herd D, numerous examinations of lungs from bacon pigs have been made but no assessment of these findings, nor an account of the unsuccessful transmission experiments made with material from the suckling pigs in herd L, is included in this paper.

Herd MG

This herd is quoted to illustrate some of the difficulties that may arise in a large herd where the litters are reared at pasture, and where there is no fattening house. It will be remembered that under the rules for herds without a fattening house, each group of pigs has to be examined as a unit, and at least 30 per cent. (now 50 per cent.) of the lungs in each group have had to be examined at slaughter.

In period 2 of the scheme, 395 lungs were examined from this herd and 182 of these had consolidated lesions in the anterior lobes, an incidence of 46 per cent. Lesions from 166 lungs were examined histologically and, of these, 4 were classified as indistinguishable, 157 were classified as suspicious, and 5 were classified as not suspicious.

The latter 5 lungs all showed alveolar collapse and

catarrh on histological examination; 3 showed small infiltrations of various cell types (mainly eosinophils), 2 showed marked interstitial fibrosis, and 1 showed very small mononuclear-cell accumulations.

The 157 lungs classed as suspicious showed part of the series of histological changes known to be associated with lung-worm infection (Dunn, Gentles & White, 1955; Dunn, 1956; Whittlestone, 1957, 1958; Mackenzie, 1958a, 1958b, 1959), and the changes seen in the present series can be summarised as follows: —

1. A bronchitis with an increase in mucous goblet cells, and infiltration of the lamina propria with mononuclear cells, but particularly with eosinophils. Lung-worms were not necessarily present.
2. A moderate or marked hypertrophy of the bronchiolar smooth muscle.
3. A peribronchitis with mononuclear-cell accumulations, but particularly with multiple nodules of proliferated lympho-reticular tissue, and sometimes with fibrosis and bronchiectasis.
4. Perivascular mononuclear-cell accumulations.
5. Moderate alveolar-cell enlargement and mononuclear-cell accumulations in the alveolar walls.
6. An accumulation in the alveoli of alveolar macrophages and multi-nucleated cells (sometimes associated with immature lung-worms or embryonated eggs), and in some cases accumulations of leucocytes (principally eosinophils) and oedema fluid.
7. Areas of vesicular emphysema.
8. Eosinophil granulomata associated with parasites or eggs.

Many of the lungs in this group showed most of the changes described above, especially the smooth-muscle hypertrophy and the lympho-reticular hyperplasia. Since enzootic pneumonia shares some of these features, however, a concurrent infection with enzootic pneumonia could not be excluded histologically and hence, on an individual basis, each lung had to be classified as suspicious.

Two of the 4 lungs classified histologically as indistinguishable from enzootic pneumonia were similar to a 2nd-stage infection (1 of them also showing a purulent bronchitis, chronic pleurisy and organisation of the interstitial tissues, and the other showing organisation and slight infiltration of the lamina propria with eosinophils, together with slight hypertrophy of the smooth muscle of two bronchioles). The third was similar to a 3rd-stage enzootic pneumonia (with a purulent bronchitis) and the last lung simulated a 4th-stage enzootic pneumonia.

It was known on several other grounds that this herd suffered heavily from parasitism, and pneumonic areas in the posterior parts of the diaphragmatic lobes were common. These were almost invariably associated with an abundance of lung-worms in the adjacent bronchioles, and lung-worms were often found similarly associated with the consolidated lesions in the anterior lobes. From a consideration of the farm as a whole, and the large groups in which the pigs were fattened, the few lesions classified as indistinguishable from enzootic pneumonia were not thought to be especially significant; they may have been but a part of the general pneumonic picture associated with widespread and heavy lung-worm infection.

In conclusion, it can be mentioned that in a herd of this type the administrative difficulties of ensuring that the requisite number of lungs from each group of pigs are examined may be considerable.

Herd Q

This herd is included to demonstrate the special diagnostic problems that can occur in herds without fattening houses. Only the 6-monthly assessment made at the end of period 2 is included, although routine lung examinations were being made before and after this time.

At the clinical inspection in December, 1959, when all the data were collated, it was found that 15 groups of pigs had been fattened. Only 5 of these, comprising 42 animals from which 33 lungs had been examined, were entirely free of consolidated lesions in the anterior lobes. The other 10 groups showed a variety of anterior-lobe lesions, and these had to be considered group by group, as epidemiological evidence is of little help in this type of herd.

Group 6 (9 pigs, 5 lungs examined) had a single contracted pneumonia of moderate extent which, histologically, showed areas of partial alveolar collapse separated by bands of scar tissue. It was, therefore, not suspicious of enzootic pneumonia, and the group was cleared.

Group 7 (14 pigs, 12 lungs examined) had a single pneumonia which consisted of 1 very small lesion of alveolar collapse and scar tissue, the perivascular and peribronchiolar tissues being infiltrated with eosinophils. It was, therefore, not suspicious of enzootic pneumonia and the group was cleared.

Group 8 (19 pigs, 13 lungs examined) had 3 pneumonias, all of which were histologically similar to the lesions seen in lung-worm infection, with parasites in 2. Since the concurrent presence of enzootic pneumonia could not be excluded histologically, they were classified as suspicious, but on over-all clinical assessment the group was cleared.

Group 9 (20 pigs, 13 lungs examined) had a single pneumonia. This very small lesion was, histologically, a typical parasitic granuloma with a chronic bronchitis. It was not suspicious of enzootic pneumonia, and the group was cleared.

Group 10 (9 pigs, 4 lungs examined) had a single pneumonia of the type described in group 8, and the whole group was again cleared.

Group 11 (14 pigs, 7 lungs examined) had a single pneumonia. Histologically, it was largely partial alveolar collapse, with capillary dilatation. As there were perivascular and peribronchiolar accumulations of lymphocytes and plasma cells in a small area, however, it was classed as suspicious of enzootic pneumonia. On general grounds the group was cleared.

Group 12 (6 pigs, 5 lungs examined) had 2 pneumonias. On histological examination, 1 was very similar to that described in group 11, and the other simulated the changes seen in lung-worm infection. Both were classified as suspicious of enzootic pneumonia, but the group was cleared.

XUM

Group 13 (12 pigs, 6 lungs examined) had a single pneumonia where the small consolidated lesion histologically closely resembled the picture seen in lungworm infection, and the group was cleared.

Group 14 (9 pigs, 6 lungs examined) had a single pneumonia which, macroscopically, resembled lesions commonly seen in enzootic pneumonia. The 3 large lesions were moist and active-looking, and histological examination revealed an alveolar-cell pneumonia with moderate accumulations of mononuclear cells around the blood vessels and bronchioles, and slight lympho-reticular hyperplasia. Thus, this pneumonia was indistinguishable from a 3rd-stage enzootic pneumonia, but there was in addition, however, moderate hyperplasia of the bronchiolar epithelium and multinucleated cells in the alveoli—features not prominent in enzootic pneumonia. No P.O. were found.

Group 15 (12 pigs, 10 lungs examined) had a single pneumonia (pig 746) which macroscopically resembled the one in group 14. Histologically, there was again an alveolar-cell pneumonia, but with more marked mononuclear-cell accumulations around the blood vessels and bronchioles, and lympho-reticular hyperplasia resulting in partial obliteration of some of the bronchiolar lumina. Some areas showed suppurative pneumonia and bronchitis, and others showed collapse. Thus, these lesions were indistinguishable from a 3rd-stage enzootic pneumonia, and P.O. were readily detected.

Groups 14 and 15 could not be safely cleared at this time, and judgment was deferred on the whole herd for a further 6-monthly period.

Herd Q is not an isolated example of this type of situation; for we have encountered a second herd (DB) with an almost identical history. Here, there was a variety of pneumonias in several groups of pigs, and among 8 pneumonias in 1 group there was 1 (pig 358) that was indistinguishable from a 2nd-stage enzootic pneumonia. As in herd Q, P.O. were detected in this pneumonia, albeit in fewer preparations, and the clinical history of the 2 pigs from which P.O. were demonstrated in herd Q and herd DB was very similar. Pig 746 in herd Q was noted to be coughing early in July, and it continued to cough periodically until slaughtered in late September; all this time it was closely housed with 11 litter-mates, from which 9 lungs examined at random were free of consolidated lesions in the anterior lobes. Pig 358 in herd DB was killed at 3 months of age because it was likewise coughing but, as before, the indistinguishable lesion was the only one of its kind in the group. From the 39 animals in this group at weaning, 24 lungs were examined; and of the 7 pneumonias other than the indistinguishable one, 6 were classed as not suspicious and the other, although classed as suspicious, was of the vegetable-material foreign-body type.

Herd TC

Although we believed from clinical and pathological observations that this herd was not free from enzootic pneumonia, it is probable that the respiratory disease problem abstracted from it was a separate entity.

Advice was sought by the farmer in November, 1958, because some litters in fold-units on an exposed site were becoming unthrifty. Because some agalactia was complicating the picture, it was not clear exactly how many litters were affected with respiratory disease, but the litters of 4 gilts were badly affected. Varying degrees of scouring occurred at 7 to 10 days of age and the pigs were seen to be checking in growth by 4 weeks of age; by 5 weeks of age their skins were waxy, dirty, pale and extensively affected with mange; in fact, the problem was presented to one of us as a skin condition. There was no coughing or sneezing in these litters at the first visit, but respiratory disease was thought to be a prime cause of the unthriftiness. Fourteen piglets were removed for further investigation and also to prevent the condition being complicated by enzootic pneumonia in the fattening stage. The survivors from the affected litters continued to grow so badly on the farm that nearly all of them were eventually destroyed.

Of the 14 animals brought to the laboratory, 4 were killed when nearly 7 weeks old, and the remainder were kept in isolation from other pigs for varying lengths of time in order to study the progressive pathology of the condition. One of the first 4 had no macroscopic abnormalities of the lung, but all the other 3 had an extensive pneumonia affecting all the lobes; in 2, the lesions were largely very pale—so much so that they resembled pancreatic tissue. Two further pigs died from fighting at 12 and 13 weeks of age, respectively; the first had no macroscopic abnormalities of the lung, but the second had a very extensive pneumonia of all the lobes. The next 2 pigs were killed at 5½ months of age; 1 had a few small areas simulating collapse, but the other had a moderately extensive pneumonia that appeared to be healing. The lesions were still largely pale, but they were now sunken and contracted. Three animals were killed at 8 months of age; only 2 showed pneumonic lesions, and these were now very shrunken, fibrotic remnants of what had probably been extensive pneumonias earlier. The latter 3 pigs were the hog pigs in a group of 6, and the remaining 3 gilts were kept for breeding by artificial insemination to see whether they would infect their litters. The only litter born was farrowed when the 3 adults were 12 months old and it was housed with its dam in a portion of the box holding the other 2 adults; hence the litter was exposed to all the 3 remaining adults during the suckling period. The dam and 1 other adult were killed at 14½ months of age, the third adult was killed at 16 months of age, and the litter of 8 were killed when they were 7 months old; all the lungs from these 11 pigs were free of macroscopic abnormalities and at no time had the litter shown clinical evidence of respiratory disease. Thus, in summary, these pneumonic lesions retained their pale character and were healing by 5½ months of age; at 8 months they were very contracted, and all 3 lungs examined after 14 months were free of lesions.

The histological picture in the 3 pigs with pneumonia killed at 7 weeks of age was consistent. In

the macroscopically pale lesions there was alveolar-cell proliferation causing thickening of the alveolar walls. The alveolar lumina contained pale-staining alveolar cells in some areas and polymorphonuclear neutrophils in others. The deeper-red lesions consisted largely of areas of partial alveolar collapse but thickened alveolar walls were seen in some areas. In both types of lesion there were a few small mononuclear-cell accumulations associated with blood vessels (and to a much lesser extent with the bronchioles) and hypertrophy of the bronchiolar epithelium. Some bronchioles contained a purulent exudate (Figs. 5 and 6).

The pneumonic lesions in the pigs killed at 3, 5½ and 8 months of age showed changes similar to those already described, except that the thickened alveolar walls contained more connective tissue elements, and the mononuclear-cell accumulations were more marked. One of the pigs 8 months old, however, had much heavier mononuclear-cell accumulations causing some obliteration of the bronchiolar lumina.

Thus, while these pneumonias showed some histological features in common with enzootic pneumonia, none closely resembled experimentally-produced enzootic pneumonia, in that there were much smaller mononuclear-cell accumulations (except in 1 of the 2 examined at 8 months), and no lympho-reticular hyperplasia; but there was obvious thickening of the alveolar walls with alveolar cells or connective-tissue elements, and more hypertrophy of the bronchiolar epithelium.

Rhinitis was not such a marked feature of this condition as in herd XI, although slight hypoplasia of the turbinates was recorded in 2 of the younger pigs (7 weeks old). Histologically, the nasal mucosae in the latter animals showed patchy mononuclear-cell accumulations in the lamina propria, neutrophil migration, proliferated surface epithelium, and some atypical glands; but no I.N.I. were detected in these pigs or in 2 others examined at 3 months of age.

Experimental transmissions. In contrast to herd XI, material taken from herd TC produced pneumonia in other pigs fairly readily. An initial contact experiment, however, was unsuccessful. Four pigs from the farm (7½ weeks old) were housed in contact with 4 other pigs (22 days old) from a herd believed to be free of enzootic pneumonia. Contact was for 3 weeks and the healthy pigs were killed 14, 25, 45 and 55 days after first mixing; none of the latter groups had pneumonia. When the 4 field cases used in this experiment were killed later, 2 of them had extensive pneumonias.

Three CD pigs (11 days old) were then sprayed with a lung suspension prepared from 2 field cases of pneumonia. They were examined *post mortem* after 10, 19 and 20 days, respectively. The youngest pig had a moderately extensive pneumonia and the other 2 had more extensive pneumonias; in all 3 animals all the lobes of the lung were affected. The pig examined after 19 days had died; it had a bilateral purulent rhinitis and it will be singled out later because I.N.I. were present. This experiment was repeated with 4 MH pigs, 20 days old. One animal was killed after 10 days; the lungs had no macroscopic abnormalities but there was a catarrhal rhinitis that was becoming purulent. The other 3 pigs were all killed after 19 days; 1 had a moderately extensive pneumonia of all the lobes and slight hypoplasia of the turbinates, but no macroscopic abnormalities were detected in the lungs and snouts of the other 2.

Further transmissions were then made using first-passage lung material harvested from the CD pigs above that were killed after 10 and 20 days. In the first of these transmissions, 2 CD pigs (8 days old) were used. One died early and the other was killed after 10 days, when it had a very extensive pneumonia of all the lobes. In the next of these second-passage transmissions, 3 CD pigs (16 days old) and 4 MH pigs (15 days old) were sprayed with the same material as had been used in the earlier second-passage experiment. One of the CD pigs soon died, and the other 2 were killed after 12 and 17 days. The pig killed after 12 days had a very extensive pneumonia of all the lobes, together with a catarrhal rhinitis and slight hypoplasia of the turbinates; the oldest pig had only small areas of pneumonia in 3 lobes, while no macroscopic abnormalities were detected in the snout. For comparison, 2 littermates of these CD pigs were sprayed on the same day with a lung suspension of the J strain of enzootic pneumonia. They were killed after 17 and 40 days, and both had developed an extensive pneumonia. The 4 MH pigs were killed after 10, 17, 25 and 37 days. The youngest pig showed several small pneumonic lesions. The next pig had only 2 very small lesions but the third had a large area of pneumonia (in 1 lobe) that was slightly contracted. The oldest pig had a single very small lesion.

To date, 1 attempt to make a third-passage transmission of this pneumonia in CD pigs (24 days old) has been unsuccessful, but there may have been a special reason for this.

The histological changes in these experimentally-produced pneumonias were as follows. On first passage, 2 of the CD pigs (killed at 10 and 20 days) showed changes very similar to those seen in the youngest field cases. No I.N.I. were detected in the nasal mucosae of these animals. The third CD pig (which had died after 19 days) and the single MH pig with pneumonia (also examined after 19 days) had different changes, in that the pneumonias were fibrinous and there was a purulent bronchitis. The MH pig had no I.N.I. in the nasal mucosa but the CD pig showed intra-nuclear inclusions (I.N.I.) in almost every gland cell of all the sections examined.

For the second passages, lung material was mixed from the 2 CD pigs that had pneumonias resembling the field cases but no I.N.I. The pneumonia thus produced in 1 CD pig after 10 days resembled the fibrinous/purulent type seen in the first-passage experiment, whereas the pneumonias produced in CD pigs after 12 and 17 days again resembled the original field disease. None of these CD pigs showed I.N.I. The same second-passage infective material sprayed to MH pigs produced 3 cases of pneumonia resembling the field disease (after

10, 25 and 37 days) and one slightly-purulent bronchitis (after 17 days). None of these MH pigs showed I.N.I.

Histologically, the 2 pneumonias produced by the J strain of enzootic pneumonia were typical of the 2nd and 3rd stages of the latter disease, and no I.N.I. were found in these animals. This parallel experiment, therefore, supported the histological differences already noted between both the naturally-occurring and experimentally-produced type TC pneumonias and the J strain of enzootic pneumonia.

Discussion

In considering the scientific lessons of the first 18 months of this control scheme, it is useful to ask two main questions. First, has enzootic pneumonia been regularly brought to light by the application of the Association's regulations and, secondly, have other diseases requiring differential diagnosis been ensnared at the same time?

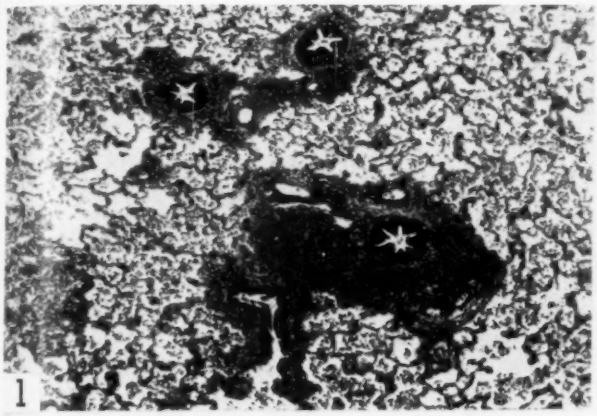
It is difficult to answer the first question at this stage, because it requires the prior establishment of a number of herds that are believed to be free of enzootic pneumonia by the present standards. No herd can claim freedom on the current regulations before July, 1960, and it will be the herds accepted at that time, together with those that come after them, that will test the hypotheses inherent in the existing rules. If enzootic pneumonia can spread only from pig to pig, it should be possible eventually to see whether the disease can lie low in a herd, evading our standards, only to emerge later. Although farm circumstances are often not reliable enough to say with certainty that there has been no contact with possibly-infected pigs, a number of break-downs in herds where such contact was highly improbable might challenge some of our assumptions. The more herds there are in the Association, the greater will be the chance of uncovering rare eventualities, such as unusual routes of transmission; the growth of the Association, therefore, is of some interest in this respect. By the end of the first year (1959), about 30 herds had satisfactorily complied with the standards. From these, new herds are being continually established, but it is too early to estimate how the number of herds claiming freedom might multiply, especially when several very different factors bear upon this.

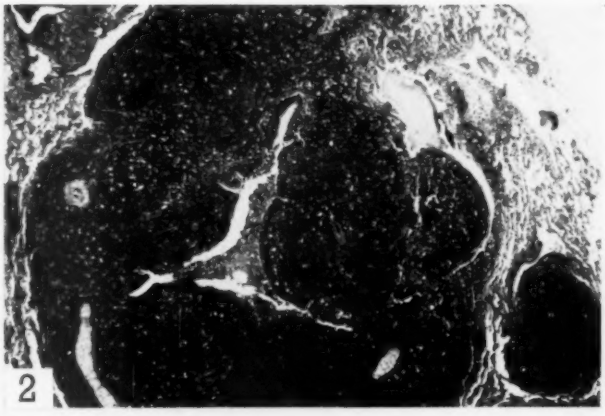
Concerning the second question, from which arise the problems of differential diagnosis, it does appear that respiratory diseases other than enzootic pneumonia are being commonly encountered. These differences may all be true ones but, on the other hand, they could, in some instances, merely indicate that our existing definition of enzootic pneumonia is incomplete. Until such time as the latter disease is defined aetiologically, and can be specifically recognised, however, these seemingly different diseases will have to be discussed in the knowledge of this limitation. Herd MG demonstrates a high incidence of anterior-lobe pneumonias where the primary aetiology—lung-worms—is in little doubt. The other diseases quoted, however, may not be so distinct aetiologically. That they could, nevertheless, be separate entities rests upon evidence of different types.

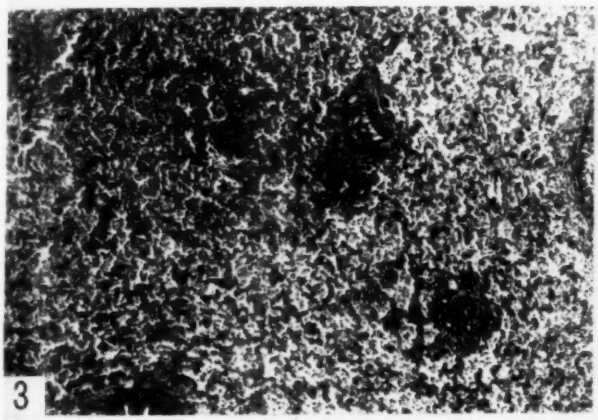
The condition seen in herd SS did not behave like enzootic pneumonia, either on the farm or in transmission experiments, even though the histological changes were very similar. Whether it is an atypical form of enzootic pneumonia, however, remains to be seen. The pneumonia from herd TC was readily transmissible to young pigs, apparently in 2 serial passages, but histologically it retained its character during transmissions and consistently differed from enzootic pneumonia. The initial disease in herds XI, L and D showed some histological differences from enzootic pneumonia (as well as being strikingly different clinically) but could not be transmitted, even though some of the material used for repeated transmission experiments was probably harvested earlier in the course of the disease than that obtained from herd TC. A puzzling feature in herds XI and L was the eventual change in the microscopic picture of the pneumonia between the time of early infection and reaching bacon weight, and this conjures the thought that enzootic pneumonia might not have a simple aetiology. That is, 2 aetiological factors might be necessary for the complete, readily-transmissible disease, either of which alone might produce a quiescent or distinctly different condition. If this were true, it might be possible to produce enzootic pneumonia by mixing together stock from herds that, individually, appeared to be free of the latter disease, but this idea is particularly speculative and will be pursued no further. The main alternative explanation, however, is that there are one or more types of pneumonia in the pig, other than that occurring in herd SS, where extensive lesions can appear, by present standards, macroscopically and microscopically indistinguishable from enzootic pneumonia. Among these, could be those described in herds Q and DB, and as pneumonias of this type could present us with some of our most difficult problems, they are receiving particular attention at the moment. Other possibilities regarding the disease in herds XI and L are, first, that the disease in the suckling pigs was clinically and histologically a different form of enzootic pneumonia, which could not be transmitted experimentally, or, secondly, that both herds became infected by extraneous sources of enzootic pneumonia before the suckling pigs reached bacon weight.

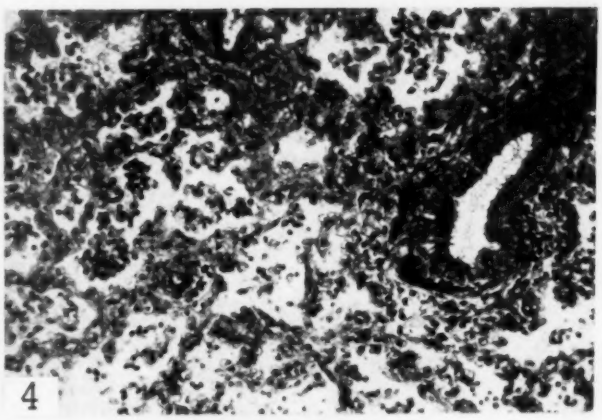
At this juncture, we prefer to offer no views on the role of the pleomorphic organisms, or of the inclusion bodies similar to those described by Done (1955), in the respiratory diseases described. An assessment of the occurrence of these pleomorphic organisms in different types of pneumonia in the field must wait upon further results. As to the intranuclear inclusion bodies occurring in the nasal mucosa, it still remains to be shown that these are specific to one disease or a group of closely-related diseases. Even if they prove to be specific, the condition with which they are associated could be so widespread that it might be transmitted concurrently with other diseases.

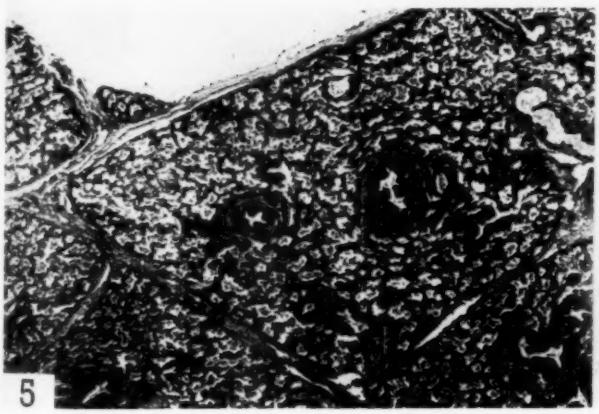
R. F. W. GOODWIN and P. WHITTLESTONE.—EXPERIENCES WITH A SCHEME FOR SUPERVISING PIG HERDS BELIEVED TO BE FREE FROM ENZOOTIC PNEUMONIA (VIRUS PNEUMONIA)











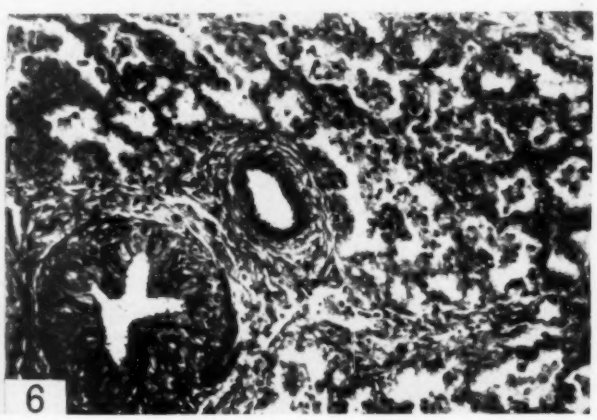


FIG. 1.— Enzootic pneumonia (3rd-stage infection). Experimental case (J strain), 38 days after inoculation. Lung showing moderate alveolar-cell proliferation and desquamation, slight thickening of the alveolar walls, moderate perivascular and peribronchiolar mononuclear-cell accumulations, some peribronchiolar lympho-reticular hyperplasia, and almost no hypertrophy of the bronchiolar epithelium. H. & E. × 50.

FIG. 2.—Pneumonia from herd SS. Field case in a bacon pig. Lung showing almost no alveolar-cell proliferation and desquamation, no thickening of the alveolar walls, and very marked lympho-reticular hyperplasia causing partial obliteration of the bronchial lumen. H. & E. × 32.

FIGS. 3 & 4.—Pneumonia from herd XI. Field case in pig 11 weeks old. Lung showing marked alveolar-cell proliferation (with some desquamation and leucocyte migration into the alveolar lumina), marked thickening of the alveolar walls in some areas, small perivascular and peribronchiolar mononuclear-cell accumulations, almost no lympho-reticular hyperplasia, and moderate hyperplasia of the bronchiolar epithelium. FIG. 3. H. & E. × 50. FIG. 4. H. & E. ×150.

FIGS. 5 & 6.—Pneumonia from herd TC. Field case in pig 7 weeks old. Lung showing marked alveolar-cell proliferation and desquamation, marked and widespread thickening of alveolar walls, almost no mononuclear-cell accumulations, no lympho-reticular hyperplasia, and moderate hyperplasia of the bronchiolar epithelium. FIG. 5. H. & E. × 32. FIG. 6. H. & E. × 150.

XUM

Our attempts to detect enzootic pneumonia might serve as a background for discussions now taking place in the pig industry on the development of so-called minimal-disease herds. As already mentioned, this term "minimal disease" seems particularly unfortunate, as it gives no idea of the diseases from which such pigs are supposed to be free. While it is possible to establish these herds under experimental conditions, the environment of ordinary farming introduces quite different factors, and it would be difficult to know to what extent the over-all disease status varied from one minimal-disease herd to the next. Once pigs begin to be transferred between herds, however, as in the scheme we describe, some knowledge must be available on the movement of disease if there is not to be a progressive deterioration in health status. Particularly, it is essential to know what agents may be present sub-clinically. In view of our difficulties with only a few respiratory diseases, the task of attempting to control all the respiratory diseases of pigs, the several forms of enteritis (of known and unknown aetiology), a range of parasitic diseases, and so on, under the ordinary conditions of transferring stock between herds, appears to be over-ambitious at this time. As far as certification of health status is concerned, therefore, we favour a policy where certain diseases that are both reasonably circumscribed and known to be of economic importance are tackled in succession. To this end, further delineation of the pig pneumonias should put the present scheme on a firmer basis and, once this is done, it might be possible to examine other respiratory diseases to see whether any of them could also be excluded from herds within the Association.

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THE SPEAKERS' INTRODUCTION

The Chairman, Dr. W. I. M. McIntyre, said that it gave him great pleasure to welcome to the Congress two distinguished workers from the University of Cambridge Veterinary School. They had worked on a large number of pig diseases and their research work was well known; in particular, they had devoted themselves to respiratory diseases of the pig, and knew more in that field between them than any other team in operation at the present time.

Dr. Goodwin was primarily a clinician and was also a research worker. He had unique experience in company with veterinary surgeons in the field in a consultative capacity in pig diseases and was that morning going to put that experience at the disposal of members.

Dr. Whittlestone was going to speak on the pathological aspect of the problem. He particularly welcomed the fact that they would, in Glasgow, see the pathological/clinical combination in operation.

Dr. Whittlestone and Dr. Goodwin were particularly anxious that the scheme which they were going to describe and the difficulties, should be clear by the end of the meeting and that questions should be freely asked on points about which there might be confusion.

Dr. Goodwin said that he and Dr. Whittlestone were very pleased to have the opportunity of explaining the comparatively small scheme that was now in operation for supervising pig herds that were believed to be free from what was commonly known as virus pneumonia. The scheme had arisen in a manner that was unusual (as far as controlling disease in this country, and perhaps in other countries, was concerned), in that it arose on the initiative of a small group of progressive farmers. This group had said that the disease was important to them in the pig industry, and that they would like to do something about it and, without any official veterinary contacts, they had got together and decided to initiate a control scheme. This meant that the veterinary profession was dissociated from it in the first instance, and it was only after the scheme had been launched for a short time that Dr. Whittlestone and he were asked to advise the Association.* They had then stipulated that they would do this only if the scheme were closely co-ordinated with the veterinary profession.

There had been a certain amount of desire from farmers to do their own lung examinations, and he and Dr. Whittlestone had not seen how it could work unless veterinary surgeons were doing these examinations. But owing to the original concept of the scheme the veterinary profession as a whole, had got rather left behind in knowing what was going on.

The paper that was being presented was a particularly long one and he felt he had to offer a number of excuses for that.

In the first place, it was really a series of papers embodying a considerable amount of field and experimental work, but, at this stage, they felt that the relationships between the different facets of their work would not be clear unless the reader were presented with all the facts together. They had, therefore, condensed what would probably be a number of later individual publications into one omnibus edition for the purpose of the Congress, in the hope that the reader would see the overall plan. The practitioner who just wanted to see how the scheme was run, however, need not read the long section on differential diagnosis.

* Dr. Goodwin was referring to the Association for the Advancement of Virus Pneumonia Free Pigs.

(*Continued on page* 1049)

APPENDIX IA PERIOD 3

THE ASSOCIATION FOR THE ADVANCEMENT OF VIRUS PNEUMONA FREE PIGS
VETERINARY SURGEON'S CERTIFICATE FOR HERDS WITH A FATTENING HOUSE

For office use only

To be completed by a Veterinary Surgeon at the herd inspection as soon as possible after 30th June, 1960, (but not later than 28th July).
This Certificate includes the results of (a) the clinical examination of the herd, (b) the naked-eye examinations of lungs that have taken place between 1st January and 30th June, 1960, (c) any microscopic examinations of consolidated lesions that were detected between 1st July and 31st December, 1959, and (d) any more recent microscopic examinations, if these are available.
Note: All periods on this Certificate are inclusive of the dates given. If you are unable to answer any question directly, or if you can supply additional information, please write explanatory notes on the back of this form. If the answer is none, write NONE.

Owner's Name and Address: .....

Name of Herd: .....

QUESTION	ANSWER	
1. Has evidence been provided by the farmer that at least one-third of each litter that reached the age of 12 weeks between 1.11.59 and 30.4.60 went into the fattening house* and remained there for 2 months?	Yes	No
2. For the period 1.1.60 to 30.6.60, has evidence been provided by the farmer that in both quarters the lungs of half the output* or of 30 pigs from the output (whichever is the lower), have been examined *post mortem* by a Veterinary Surgeon?	Yes	No
3. What was the output of the fattening house (a) between 1.1.60 and 31.3.60?	(a)	
(b) between 1.4.60 and 30.6.60?	(b)	
4. How many lungs* were examined (a) between 1.1.60 and 31.3.60?	(a)	
(b) between 1.4.60 and 30.6.60?	(b)	
5. How many lungs had consolidated lesions of the anterior lobes* (a) between 1.1.60 and 31.3.60?	(a)	
(b) between 1.4.60 and 30.6.60?	(b)	
6. If between 1.1.60 and 30.6.60 more than 5% of lungs had consolidated lesions of the anterior lobes, have lesions from all these affected lungs been despatched to the Animal Health Trust, Stock?	Yes	No

7. Between 1.1.60 and 30.6.60 (recording each quarter separately)		1.1.60 to 31.3.60	1.4.60 to 30.6.60
(a) from how many lungs have specimens been sent for microscopic examination?	(a)	.....	.....
(b) according to the reports **already available,** how many lungs have been classified as:			
(i) not suspicious of virus pneumonia?	(b) (i)	.....	.....
(ii) suspicious of virus pneumonia?	(ii)	.....	.....
(iii) indistinguishable from virus pneumonia?	(iii)	.....	.....
8. Between 1.7.59 and 31.12.59			
(a) from how many lungs were specimens sent for microscopic examination?	(a)	.....	.....
(b) how many lungs were classified as:			
(i) not suspicious of virus pneumonia?	(b) (i)	.....	.....
(ii) suspicious of virus pneumonia?	(ii)	.....	.....
(iii) indistinguishable from virus pneumonia?	(iii)	.....	.....

9. How much coughing is there in the fattening house?	None	Some	Much
10. Is there any clinical evidence of respiratory disease among the pigs that are not in the fattening house?		Yes	No
11. Between 1.1.60 and 30.6.60 has there been, to your knowledge, any respiratory disease among the pigs that is no longer apparent?		Yes	No

I certify, to the best of my knowledge and belief, that the above answers are correct.

Date..... Signed .....
(Veterinary Surgeon)

Name of the Veterinary Surgeon who has examined the lungs, if different from the above: .....
(Veterinary Surgeon)

* See standards for definitions.

THE ASSOCIATION FOR THE ADVANCEMENT OF VIRUS PNEUMONIA FREE PIGS PERIOD 3

FARMER'S DECLARATION FOR HERDS WITH A FATTENING HOUSE For office use only

To be completed by the owner or agent as soon as possible after 30th June 1960 (but not later than 28th July).

All the statements in this declaration relate to the period 1.1.60 to 30.6.60 inclusive and they must all be answered. If the answer is none, write NONE.

1. Pigs from this herd have gone only to the herds listed below.

Herd	Type of Pigs*	Dates of Delivery
.....	.....	.....
.....	.....	.....
.....	.....	.....

2. Pigs introduced into this herd have come only from the herds listed below.

Herd	Type of Pigs*	Dates of Arrival
.....	.....	.....
.....	.....	.....
.....	.....	.....

3. No pigs have left or strayed from this herd and returned, except those listed below.

Animal	Details of Incidents
.....	.....
.....	.....
.....	.....

4. Apart from the exceptions listed in questions 2 and 3 above, to the best of my knowledge, the only possible contacts† between this herd and pigs of other herds are as follows.

Details of Possible Contacts

.....

.....

.....

5. Is the fattening house† in this herd a building in which the main living quarters for the pigs are largely enclosed and where there is free interchange of air between all the pens? Yes No

6. Apart from the exceptions listed below, the fattening house has at no time between 1.1.60 and 30.6.60 contained less than 20 pigs.

Dates of Exceptions	Details

7. The output† from the fattening house between (a) 1.1.60 and 31.3.60 was .....

(b) 1.4.60 and 30.6.60 was .....

8. Have you any reason, apart from those given in the answers above, to suspect that virus pneumonia is present in this herd? Yes No

I certify, to the best of my knowledge and belief, that the above answers are correct.

Date: ..... Signed ..... (Owner/Agent).

Name of Herd: ..... Address: .....

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* State whether boars, gilts, weaners, etc. The numbers involved are not required.

† See standards for definitions.

PERIOD 3
For office use only

APPENDIX 1B

THE ASSOCIATION FOR THE ADVANCEMENT OF VIRUS PNEUMONIA FREE PIGS

VETERINARY SURGEON'S CERTIFICATE FOR HERDS WITHOUT A FATTENING HOUSE

To be completed by a Veterinary Surgeon at the herd inspection as soon as possible after 30th June, 1960, (but not later than 28th July).
This Certificate includes the results of (a) the clinical examination of the herd, (b) the naked-eye examinations of lungs that have taken place between 1st January and 30th June, 1960, (c) the results of any microscopic examination of consolidated lesions that were detected between 1st July and 31st December, 1959, and (d) any more recent microscopic examinations, if these are available.
Note: All periods on this Certificate are inclusive of the dates given. If you are unable to answer any questions directly, or if you can supply additional information, please write explanatory notes on the back of this form. If the answer is none, write NONE.

Owner's Name and Address: .....

Name of Herd: .....

QUESTION	ANSWER
1. For the groups fattened* between 1.1.60 and 30.6.60, is there evidence that	
(a) when weaners have been mixed in groups of 3 or more litters the lungs from 30% of the pigs that started in each group have been examined *post mortem*, at least 2 months after mixing, by a Veterinary Surgeon?	Yes No
and	
(b) the lungs of 50% of the numbers weaned in each litter but not mixed in groups of 3 or more litters, have been examined *post mortem* by a Veterinary Surgeon?	Yes No
2. Between 1.1.60 and 30.6.60 have specimens from all lungs showing consolidated lesions of the anterior lobes* been sent to the Animal Health Trust, Stock?	Yes No

3. For the periods shown on the right, how many lungs—			1.7.59 to 13.12.59	1.1.60 to 31.3.60	1.4.60 to 30.6.60†
(a) were examined?	(a)		.....	.....	.....
(b) had consolidated lesions of the anterior lobes?	(b)		.....	.....	.....
(c) had pneumonic lesions microscopically					
(i) not suspicious of virus pneumonia?	(c)	(i)	.....	.....	.....
(ii) suspicious of virus pneumonia?		(ii)	.....	.....	.....
(iii) indistinguishable from virus pneumonia?		(iii)	.....	.....	.....

4. How much coughing is there in			
(a) unweaned piglets?	None	Some	Much
(b) fattening pigs?	None	Some	Much
(c) adult pigs?	None	Some	Much

5. Between 1.1.60 and 30.6.60, has there been, to your knowledge, any respiratory disease among the pigs that is no longer apparent?	Yes	No

I certify, to the best of my knowledge and belief, that the above answers are correct.

Date: ..... Signed: .....
(Veterinary Surgeon)

Name of the Veterinary Surgeon who has examined the lungs, if different from the above.

Name: .....
(Veterinary Surgeon)

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* See standards for definitions.
† Enter the results of any reports available.

THE ASSOCIATION FOR THE ADVANCEMENT OF VIRUS PNEUMONIA FREE PIGS

FARMER'S DECLARATION FOR HERDS WITHOUT A FATTENING HOUSE

PERIOD 3

For office use only

To be completed by the owner or agent as soon as possible after 30th June 1960 (but not later than 28th July).

All the statements in this declaration relate to the period 1.1.60 to 30.6.60 inclusive and they must all be answered. If the answer is none, write NONE.

1. Pigs from this herd have gone only to the herds listed below.

Herd	Type of Pigs*	Dates of Delivery
.....	.....	.....
.....	.....	.....
.....	.....	.....

2. Pigs introduced into this herd have come only from the the herds listed below.

Herd	Type of Pigs*	Dates of Arrival
.....	.....	.....
.....	.....	.....
.....	.....	.....

3. No pigs have left or strayed from this herd and returned, except those listed below.

Animal	Details of Incidents

4. Apart from the exceptions listed in questions 2 and 3 above, to the best of my knowledge, the only possible contacts† between this herd and pigs of other herds are as follows.

Details of Possible Contacts

5. Have you any reason, apart from those given in the answers above, to suspect that virus pneumonia is present in this herd? Yes No

I certify, to the best of my knowledge and belief, that the above answers are correct.

Date: ..... Signed: ..... (Owner/Agent)

Name of Herd: ..... Address: .....

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* State whether boars, gilts, weaners, etc. The numbers involved are not required.

† See standards for definition.

APPENDIX 2

STANDARDS FOR HERDS CLAIMING FREEDOM FROM VIRUS PNEUMONIA

REVISED MARCH 10th, 1960

1. (a) The requirements listed in the following paragraphs must have been fulfilled for a period of at least 18 months

OR

(b) the herd must (i) have been founded, without any contact* with possibly infected pigs, only from other herds whose freedom from virus pneumonia has been established according to the Standards of the Association, and (ii) have itself complied with the Association's Standards for a period of 6 months, any time after the birth of the first litter in the new herd.

2. **LUNG EXAMINATIONS FOR HERDS WITH A FATTENING HOUSE***

(i) At least one-third of each litter must have gone into and remained in the fattening house for a period of 2 months in accordance with the requirements of the Veterinary Surgeon's Certificate.
(ii) In each quarter, the lungs* of half the output,* or of 30 pigs from the output (whichever number is the lower), must be examined *post mortem* by a veterinary surgeon.
(iii) In each period of 6 months
either (a) at least 95 per cent of the lungs examined must be free from consolidated lesions of the anterior lobes*
or (b) when 5 per cent or more of the lungs examined have consolidated lesions of the anterior lobes, lesions from all these affected lungs must be examined microscopically and 75 per cent or more of the affected lungs must then be free from suspicion of virus pneumonia *or*, if they are not free from suspicion to this extent, an approved consultant, after an investigation of the situation, must declare the herd to be free from virus pneumonia.

LUNG EXAMINATIONS FOR HERDS WITHOUT A FATTENING HOUSE*

(i) Weaners must be mixed in groups of 3 or more litters and the lungs from at least half the pigs that started in each group be examined *post mortem*, at least 2 months after mixing, by a veterinary surgeon, OR
if the weaners are not mixed in groups of 3 or more litters,
either (a) when the age at slaughter is more than 18 weeks, the lungs of at least two-thirds of the pigs weaned in each litter must be examined *post mortem* by a veterinary surgeon
or (b) when the age at slaughter is between 14 and 18 weeks, the lungs of at least half the pigs weaned in each litter must be examined *post mortem* by a veterinary surgeon.
(ii) In any of the cases of (i) above, all the lungs must be free from consolidated lesions of the anterior lobes, or lesions from all lungs showing consolidation of the anterior lobes must be examined microscopically and the status of each herd will be assessed, if necessary by an approved consultant, on the basis of the results of the microscopic examinations together with any additional information that may be required.

3. **HERD INSPECTIONS**

Every 6 months the herd must be inspected by a veterinary surgeon appointed by the farmer, and the Veterinary Surgeon's Certificate and the Farmer's Declaration must be completed and returned to the Association.

4. There must be no contact* between the herd and possibly infected pigs, except under the supervision of the Association. A herd wishing to be supervised in this respect must first notify the Association of its intentions, have its plans approved or modified, and complete the plans to the satisfaction of the Association.

5. Herds will be classified either as having complied with all the Association's Standards, or as being under supervision. There will be a further distinction between herds with and without fattening houses. In all cases, introduction of pigs from a herd of lower status results automatically in the transfer of this lower status to the recipient's herd.

6. The Standards may be modified at a General Meeting in the light of experience.
NOTE.—Compliance with the Standards of the Association carries no guarantee that a herd is free from virus pneumonia.

* DEFINITIONS

The following terms have a special meaning in the Association:

1. A **contact** must be recorded as occurring when pigs remaining in the herd
   (a) come within 10 yards of possibly infected pigs, even momentarily,
   (b) are ever in a building with possibly infected pigs,
   (c) are continually kept within 50 yards of possibly infected pigs,
   (d) occupy premises that have housed possibly infected pigs within the previous 14 days, or
   (e) are moved in transport that has
      (i) not been disinfected by an "approved" disinfectant and then left empty for 12 hours, or
      (ii) housed possibly infected pigs within the previous 14 days.

2. A **fattening house** is a building in which the main living quarters for the pigs are largely enclosed and where there is a free interchange of air between all the pens. It must never house less than 20 pigs and no quarterly output should be less than 40 pigs. If the last two requirements have not been met, the herd will require special consideration by the Association.
Note:—When the one-thirds of every litter do not remain in a single fattening house for 2 months, the regulations governing fattening houses and lung examinations must be applied individually to each of the houses that together house these one-thirds.

3. A **lung** is all the lung tissue from one pig.

4. The **output** is the number of suckling or fattening pigs that have remained in the fattening house for at least 2 months and then been removed for any reason, (e.g. because they have died, been slaughtered or kept for breeding).

5. The **anterior lobes** are those parts of the lung anterior to a line joining the widest parts of the diaphragmatic lobes.

Secondly, it was often as important to see the reasons behind actions as to explain the actions themselves, and this was particularly so with the control scheme being described, because their views on respiratory disease in the pig were rather different from those expressed previously or commonly stated elsewhere. In the first part of the paper, they had taken some trouble to try to explain how they were thinking about enzootic pneumonia of pigs. Their joint views on the question could best be summarised by saying that they saw the respiratory disease position in pigs as being rather similar to that in chickens, except that in the latter species the problem had been more clarified, in so far as more individual diseases had been recognised and differentiated within the total respiratory complex. Before embarking on any control scheme for enzootic pneumonia, therefore, they had been quite prepared for the existence of a number of different infectious pneumonias in pigs; some of those would probably turn out to be primarily pneumonic in type, whereas others would be secondary to infections more specific to the upper respiratory tract or perhaps the bronchi. Thus, eventually, they might be able to classify those respiratory infections as different forms of rhinitis, different forms of pneumonia, and different types of bronchitis. This was not a view that one met commonly elsewhere; for the tendency was to speak of virus pneumonia and atrophic rhinitis as if these were two distinct entities in pigs which, between them, embraced almost all the ills of the respiratory tract. Arising from that, they were perplexed by control schemes in operation abroad. They had had some discussion with veterinary surgeons who were operating parallel schemes, and Dr. Whittlestone had returned from Sweden only last week, but that aspect might be returned to in the discussion.

The third reason for the length of their paper was because they wished to explain to practitioners the detailed workings of the scheme now in progress, so that if they were asked to co-operate by farmers, they would know in advance what was involved. As the Congress served mainly to help practitioners, he would explain as briefly as possible the essence of the methods that had been adopted to detect the presence of enzootic pneumonia in a herd. In stressing the field side of the paper, however, it did not mean that they would not be equally willing to deal with any questions of a more specialised nature, in due course, on the experimental results presented.

The regulations devised for detecting enzootic pneumonia might be regarded as a net. That net, which had been designed from their previous knowledge of the epidemiology of the disease, was continually trawled through all the herds that wished to claim freedom from enzootic pneumonia in the Association for the Advancement of Virus Pneumonia Free Pigs. At that point, it could be interjected that, when talking of enzootic pneumonia, virus pneumonia was meant as far as the Association was concerned but, scientifically, they believed that their definition of enzootic pneumonia was a more limited concept than had hitherto been included in the term virus pneumonia. In other words, a number of pneumonias that they regarded as different from enzootic pneumonia had, in the past, all been grouped together as virus pneumonia of pigs.

It was also mentioned in the paper that they were not too happy about giving the name virus pneumonia to a disease until it was shown with certainty that it was caused by a virus. Therefore they used the term enzootic pneumonia, which to the best of their ability they were trying to confine to one condition.

The operation of the net was based on a few important assumptions, which were best illustrated when the herd was one with a fattening house. There were regulations for herds without a fattening house about which they had never been very happy, but these latter herds were accorded a different status in the Association from those with fattening houses, even though they appeared to be free of enzootic pneumonia.

The assumptions being made were that the cause of enzootic pneumonia could not exist in a closely-housed population of pigs without (a) causing pneumonia and (b) causing the incidence of pneumonia in the anterior lobes to exceed the 5 per cent. level.

The whole basis of the scheme depended on the detection of pneumonia. If it were later found that it was possible for the cause of enzootic pneumonia to tick over in a herd without causing any pneumonia at all, the way in which the disease was now detected fell to the ground. There was no evidence so far, however, that that occurred.

With regard to the second assumption, the anterior lobes were defined in the Association as being anterior to the widest part of the diaphragmatic lobes. Therefore, to determine whether the causal agent was present in a herd, each litter was sampled. This was done by taking a sample of pigs from every litter born (the regulations said at least one-third of each litter) and mixing them together for long enough to spread the disease (the regulations said two months). Providing the pigs were continually fed into the bottom end of the production line, the disease, if present, should build up to a point beyond the minimum safety level; and to allow sufficient time for the build-up (as when there might be only one or two intermittent carriers in the sows), the regulations said that the litter sampling and lung checking had to continue for 18 months before the Association would first accept the herd as being free. The presence of the disease was shown by the examination of a stipulated number of lungs from the output of the fattening house every quarter by a veterinary surgeon. It was important to note that they were not prepared to accept the opinion and certificate of a non-veterinary person, such as a lay meat-inspector.

The second main assumption was that the disease spread mainly from pig to pig through droplet infection. If a herd was shown to be free of the disease, it should remain free; unless it came into contact with infected pigs. Hence contact was rigidly defined both as regards time and distance.

The net, designed to catch enzootic pneumonia, would also catch any other respiratory disease with an incidence of consolidated lesions in the anterior lobes of the lung of more than 5 per cent. In that event, and as a first screening test, histological examinations of the lesions were made. If that failed to give a definite lead, the whole position had to be investigated anew and further tests carried out if necessary.

Although they had written at some length on enzootic pneumonia, at no point had they expressed any views on the economic effects of that disease or on the relative economic effect of other respiratory diseases in the pig. He was collecting information on that aspect at the moment and had definite views on the effects of both enzootic pneumonia and other respiratory diseases in many herds under a wide variety of conditions; but as far as the paper was concerned, they were confining themselves to scientific problems of differential diagnosis, which were every bit as real whether the diseases that were confusing the issue had any economic effect or not.

Another point was the existence of respiratory infections in herds said to be free of enzootic pneumonia. There had been some criticism of their attitude here, and they had been accused of only partly doing the job. They found that criticism difficult to understand, unless those who made it still believed that all pneumonias in the pig were a single disease. One did not hear similar criticism of other control schemes, and if the Ministry of Agriculture set out to control swine fever, they could not be legitimately criticised for not controlling other diseases at the same time. In the authors' view, to include other diseases that were superficially similar was a never-ending road to ruin. Only in the most exceptional circumstances could they see any future in trying to control a disease condition that had a very hazy definition. It was possible, for example, that the Atrophic Rhinitis Order was instituted because it was felt that a disease called atrophic rhinitis had recently entered the country from abroad. A case could be made, therefore, for a swift and complete attack on it; but the great difficulties of operating such an Order against ill-defined clinical signs were clear to-day. As explained in the paper, they had taken on the task of advising the Association on so-called virus pneumonia with some trepidation, and at this stage they were not prepared to consider the question of controlling all or most of the respiratory diseases of pigs which, from previous experience and a moment's reflection, should

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appear to most people as both scientifically and practically untenable.

When it came to question time it might be useful to know where the responsibility of the two authors divided. He was responsible for all the field work, for all the macroscopic lung examinations arising from that work, and for making the final decision in difficult herds after compiling all the laboratory and field evidence. Dr. Whittlestone was responsible for all the microscopic and cultural work. They carried out experimental studies together. Dr. Whittlestone, in his opening remarks, would deal with the laboratory side; he would also touch upon the experimental transmissions, because he had had extensive experience of the experimental behaviour of enzootic pneumonia before their joint studies began on the wider respiratory disease complexes of the pig.

Dr. Whittlestone said that in his introductory remarks he would like to indicate the part that the various laboratory methods played in the recognition of enzootic pneumonia. At the moment, there were no means of identifying the causal agent except with regard to its general characteristics, nor had any individual specific features been recognised in the disease. Thus the laboratory methods, although helpful, played only a limited part in the recognition of the disease. He would confine his remarks, therefore, to three aspects of the laboratory methods which did appear to be of use: firstly, histology of consolidated lesions in the lungs; secondly, examination of touch preparations (impression smears) of pneumonic lung for pleomorphic organisms; and thirdly, experimental transmissions.

With regard to the part played by histology, as Dr. Goodwin had pointed out, in herds with a fattening house, if the incidence of consolidated lesions of the anterior lobes exceeded 5 per cent., then pneumonic tissue from all the lungs was examined histologically.

Dr. Goodwin had also indicated that they thought of the respiratory affections of the pig as a disease complex, and by analogy with the respiratory disease position in other species, and from the information already available in the pig, they would expect a number of different primary and secondary agents, as well as different predisposing factors, to be involved in the production of the commonly recognised lesions (*e.g.* pneumonia and turbinate hypoplasia). The histological picture which occurred in lungs affected by enzootic pneumonia was the sort of picture that might well occur as a result of infection with several different agents. A certain amount of evidence that pneumonias behaving differently epidemiologically could not be distinguished from enzootic pneumonia histologically was already available before the Association was launched, and hence little hope existed for a specific histological diagnosis. With the growth of the Association, and the consequent greatly increased study of respiratory diseases apparently behaving differently from enzootic pneumonia, that limitation on the value of histology became much more striking. An increasing number of pneumonias were being encountered that shared important histological features with enzootic pneumonia, but appeared to differ from that disease in a number of other ways, *e.g.* epidemiologically and in their transmissibility.

It might have been noticed that in the Association's forms, the words histologically "suspicious of" or "indistinguishable from" enzootic pneumonia were used. He stressed that those words carried no implication that a pneumonia so classified was enzootic pneumonia. He mentioned that point because a number of people still thought that those words implied that diagnostic histological features were known in enzootic pneumonia. That, unfortunately, was not the case. The remarks he had just made on the limitations of histology in diagnosing enzootic pneumonia applied, of course, to the situation as it now stood. It was always possible that more detailed studies would reveal features more characteristic of enzootic pneumonia.

He had indicated the limitations of histology in differentiating the pig respiratory diseases. But histology was really useful in those instances when the consolidated lesions showed histological pictures distinctly different from enzootic pneumonia, particularly if the respiratory condition could be differentiated from enzootic pneumonia on other grounds as well.

Even in those instances where pneumonias shared important histological features in common with enzootic pneumonia, the histology could still be of use, particularly when many lungs were examined from a herd. That was because small differences became more significant with large numbers.

Finally, the histological opinion became more valuable when the histological findings could be related to the epidemiological behaviour of the pneumonia.

With regard to the part played by the examination of touch preparations (impression smears) for the Pleomorphic Organism (P.O.), it had been indicated in the paper, and elsewhere, that very small delicate pleomorphic organisms of the right size to be the causal agent could be found in touch preparations of experimentally-produced enzootic pneumonia. To date, P.O. had readily been found in the few herds that they believed to be infected with enzootic pneumonia according to their latest criteria, which had been described in the paper. They had also described in the paper, some respiratory diseases that, although resembling enzootic pneumonia histologically, had not presented P.O. in smears, and it was interesting that those diseases had differed from enzootic pneumonia in other respects. However, to make the position more complicated, structures appearing quite similar to P.O. had been found in some pneumonias that had behaved differently from enzootic pneumonia. Since P.O. were identified only morphologically at the moment, it was not known whether the P.O. observed in different outbreaks of pneumonia were the same organism or not. If there were several types of P.O., it would be interesting to know whether one particular type of P.O. was specific for enzootic pneumonia. As they had said in the paper, they hoped that further studies, which were now in progress, would cast more light on the role of P.O. in enzootic pneumonia and other respiratory affections.

As to the part played by experimental transmissions, all the strains of enzootic pneumonia studied experimentally had readily produced pneumonia in unweaned or weaned pigs, and those experimentally-produced pneumonias had been contagious to other pigs. There had been described in the paper a respiratory disease which appeared to be infectious in the field, but which differed from enzootic pneumonia epidemiologically. That disease, however, could not be transmitted experimentally as a pneumonia to other pigs—even to very young, colostrum-deprived animals, which might have been expected to be very susceptible. It was possible, therefore, that the facility with which a pneumonia could be transmitted experimentally could be of considerable help in diagnosis. It was not known, however, whether enzootic pneumonia from the field was, in all instances, infective experimentally.

Some other pneumonias described were transmissible experimentally, but continued to differ from enzootic pneumonia in their behaviour in the experimentally-infected pigs; that type of information was of considerable help in classifying the various conditions.

Thus, although each of the methods used had its limitations, all the epidemiological and laboratory evidence together was of considerable value in differentiating the various respiratory diseases being studied.

Dr. J. K. Walley (Knutsford), said that he was interested in the 5 per cent. tolerance level. Were the consolidations ever indistinguishable from the lesions in which the authors were interested, and had they ever taken from those consolidated portions material which was transmitted and which had been indistinguishable from enzootic pneumonia?

Dr. Goodwin, in reply, said that it was very rare to get pneumonia simulating enzootic pneumonia, completely, at less than the 5 per cent. incidence. The course Mr. Walley suggested would be taken, but there were so few opportunities; they were now in the middle of doing it. There was one such case of pneumonia in both herd Q and herd DB, as described in the paper; these pneumonias were indistinguishable histologically from virus pneumonia but did not spread in the herd. Obviously, pneumonias of that type were of considerable interest, because they tested the hy-

pothesis of whether it were possible for the causal agent to exist undetected. It was worth while pointing out that both these herds were without a fattening house. They had always been unhappy about the situation that had grown up in the early days of the Association concerning the admission of herds without fattening houses. Perhaps one day, such herds might be removed from the Association's scheme. He did not think there was evidence at the moment that, under intensive conditions, the situation mentioned by Mr. Walley arose.

Dr. Walley asked what happened in fattening houses with pneumonias which, very frequently, were not pneumonias at all but were collapse, or sometimes, there was pneumonal inhalation of vegetable material. Frequently, there was pneumonia of the anterior lobes associated with worm infection.

Mr. W. H. Parker (Wolverhampton) said that it seemed that the practical side was important. He felt that because every now and then, a request was received at the laboratory to start examining lungs; they were expected to run out to some abattoir two or three times a week! Who was supposed to examine the lungs of herds in the scheme? How was there a link-up between examination of lungs macroscopically and the examination of the over 5 per cent., if any, histologically? Which laboratory did it? He presumed it was the Cambridge one.

Dr. Goodwin, in reply, said that there had been administration problems arising from that. As explained in the paper, he himself had often done the work normally carried out by practitioners, so that he had been through the practical difficulties in the field. Working the scheme locally was not as difficult as some people believed. If the regulations were read, it would be seen that, for a herd with a fattening house, the veterinary surgeon had first to examine the lungs of only 30 fat pigs each quarter. If the work were arranged and thought about, and discussed with the farmer (and with most farmers this was an important thing if they wanted to sell virus-pneumonia-free breeding stock) it would not be a burden to examine lungs in this way. Then a six-monthly inspection had to be carried out on the farm. As to how it was done, the certificates were submitted by the practitioner. A farmer usually bought a herd from another man who already had a virus-pneumonia-free herd, and then said that he wanted to see that his own herd remained free from virus pneumonia, so that he, in turn, could sell virus-pneumonia-free-pigs and establish further herds. The farmer should approach his practitioner, after getting the necessary papers from the Association, and they should go through them together. It often appeared, however, that the practitioner had not read the regulations in advance to see what was involved. Arrangements could then be made for the lungs to be examined. If the pigs were slaughtered locally, the practitioner could arrange to go to the local slaughterhouse—the lungs could be put in the refrigerator overnight. If, on the other hand, the farmer had a contract with a more distant bacon factory, arrangements should be made with another practitioner to carry out the lung examinations. Allowance was made for that on the certificates. Obviously there had to be co-ordination between them. Many lung examinations, for example, were being made at Harris's factory in Wiltshire, which had a qualified veterinary surgeon as meat inspector. This inspector was reporting back the results to the individual local practitioners, who did the final co-ordinating for the herd certificate.

With regard to what happened if there were consolidated lesions, all consolidated lesions should be recorded. This was very important. Now that the certificates were coming in, many veterinary surgeons were saying, "In my opinion, there are no lesions of virus pneumonia." They were not asking the practitioner, however, to say whether he thought the lungs had virus pneumonia or not; all they asked him to say was whether there were any consolidated lesions of any sort. The rest would be sorted out centrally. If lesions were found, the practitioner could not usually know whether he was going over the 5 per cent. level or not; so he had to retain the lesions in formol-saline until he saw the final incidence of lungs with lesions. Once the 5 per cent. incidence had been exceeded, histological examinations had to be done. A laboratory had to be found, therefore, that would carry out this work. The Animal Health Trust at Stock agreed to provide this service for a standard charge; they had been at the early discussions and it had been accepted that, histology being such a subjective subject, there could not be many laboratories doing the histological reporting, because there would have to be an agreed policy as to what was to be classified as indistinguishable from, suspicious of, and not suspicious of virus pneumonia.

In these early days of the Association, there were some inevitable administrative difficulties: sometimes, for example, the co-ordination broke down between the veterinary surgeon doing lung examinations at the bacon factory and the veterinary surgeon who should be co-ordinating everything on the farm, in that it had not been made clear whether the farmer had a fattening house or not.

Most of the farmers who were in the scheme seemed to be in the progressive section of the pig industry; he got the impression that they were taking it very seriously and that it meant a lot to them. It could bring an advantage in the sale of virus-pneumonia-free gilts and boars, and it was worth while realising that often much was at stake. The clinical inspection, for example, could not be tossed off in five minutes. He did a number of these inspections on behalf of practitioners who had not wanted to do it themselves, and he had found that it took him the whole afternoon to correlate all the many examinations for the previous six months, make sure all the required things had been done on the farm (such as a third of each litter going into the fattening house), and to sign the certificate. At the end, the certificate should mean something for anyone who wanted to buy stock.

If the regulations were sound, they could also be used to see whether any herd of unknown status had virus pneumonia or not. There would probably be few herds without virus pneumonia, although occasionally a herd was free of virus pneumonia by chance. If a farmer came and said: "Have I got virus pneumonia or not?"—and if he had a fattening house he usually knew he had got it when he came—the first thing to do was to examine 30 to 40 lungs and see whether the incidence of affected lungs was well over the 5 per cent. level. If they were, then they should be put through for histological examination, and if they were all indistinguishable from virus pneumonia, in most cases the herd would not be virus pneumonia free. Concerning the various methods by which a farmer could gain a virus-pneumonia-free herd, he could either eradicate it from his own stock in a variety of ways, or buy a new herd from someone who already had one. The Association was more or less a certificate-issuing Association, trying to regulate the unsatisfactory position that had grown up four or five years ago when people were inserting advertisements in the farming press about "virus-pneumonia-free pigs", although few farmers really knew what a virus-pneumonia-free pig was or how to test the respiratory disease status of their herd.

A Member asked a question which he said was in a lot of people's minds. How much had to be paid for it? Who paid for the examination? Did the farmer pay it or did the Association pay it? Was there any idea, from the 30 odd farmers who were in the scheme now, how much per pig it cost them to be in the scheme?

Dr. Goodwin said that the scheme was entirely private and self-supporting. The Association did nothing but administer. It had no responsibility for what would be equivalent to testing in the attested herds scheme. The subscription to the Association was five guineas a year, and there was an associate subscription for veterinary surgeons of 10/- a year. For that, all the literature of the Association was available, together with the regulations and the newsletter on general progress. When all the regulations had been conformed with, the herd's name could be published in the Association's list, and it was hoped that the regulations would be so strict that the list would come to mean something among pig farmers. Provided everybody had done their job, the Association's list should only have on it those herds that were free from virus pneumonia.

The practical question of complying with the standards was purely an arrangement between a farmer and his veterinary surgeon. The only thing the farmer paid officially

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to the Association was his subscription and a charge to have his name published on the list of qualified herds. The farmer would normally go to his veterinary surgeon and say, "Will you help me to get a virus-pneumonia-free herd? Will you carry out the lung examinations?" If the veterinary surgeon agreed to do this, he presumably charged what he thought his time was worth for doing work that should be of considerable value to the herd per year.

As to what it would cost the farmer, in all, he did not know what practitioners were charging for this service; but what it was worth to the pig farmer and what he got out of it had been touched upon by Professor Beveridge earlier in the week in the paper on the economics of animal health. When the original experimental work at Cambridge was made some years ago, pigs fattened with and without virus pneumonia, showed a difference in conversion ratio of about 20 per cent.

He had seen many herds, under a great variety of conditions, some with a number of respiratory diseases, and many with enzootic pneumonia. The effect of any disease was very variable, of course. He knew of some herds, for example, where the incidence of pneumonic lesions at slaughter was running at about 95 to 100 per cent., and those herds were completely uneconomic. There were all gradations down from this severity; he supposed the average was about 45 to 50 per cent. incidence of pneumonia at slaughter.

In summary, freedom from virus pneumonia was worth three things. First, each affected pig had got to eat more food (according to how badly affected it was) to get to the bacon factory. Overall, there might be a difference of 20 per cent. in the herd conversion ratio, which was a considerable amount of money a year. Many progressive farmers had said that it would be well worth being virus-pneumonia-free, being a member of the Association, and paying their related veterinary fees, for the straight economic benefit of getting their pigs to the factory more quickly—quite apart from the extra bonus of selling breeding stock to other people.

Secondly, enzootic pneumonia was a disease very much like tuberculosis in cattle, when practitioners used to say, "Now we have this herd free of tuberculosis, we notice the whole health is better." This effect of a chronic disease on the general health of a population must have been experienced by many veterinary practitioners.

The third benefit was related to the fact that the biggest people were thinking of producing pigs like broilers; it was becoming more intensive. Factory methods were becoming more important. Mr. Johnson at Lydney, for example, used to fatten 54 pigs every week; he rarely fattened 53 or 55—just like seeing motor cars coming out of Morris Cowley Works. He said that one cannot do intensive pig production in the modern manner unless everything was streamlined; if a chronic disease of this type is present it meant all your production belts were working at different rates. In one period of 13 consecutive weeks in a quarter, taking all the 54 pigs going off every week together, every pig of the 700 went to the bacon factory within plus or minus three days of the average figure. Such a regularity of performance could not be achieved in a herd of that size if enzootic pneumonia existed in the pigs.

In answer to a question as to whether the certificate of the Association was accepted by any foreign countries or export markets, Dr. Goodwin replied that it was not as far as they knew.

Mr. Mackenzie asked two questions on the economic advantages of being a member of the Association. Was it found, on the initiative of veterinary surgeons, that some pig farmers were eradicating virus pneumonia from their herds without becoming members of the Association?

On the experience obtained to date, was there any likelihood of bacteriological pneumonia becoming more serious in those herds which were encouraged to be crowded together in the fattening house?

Dr. Goodwin said it was quite possible for a man to have a virus-pneumonia-free herd without having anything to do with the Association. As time went on, as he saw it, there would be an increasing number of people who would keep virus-pneumonia-free herds purely for commercial reasons; they would hope they stayed free and would carry out the Association's recommendations and follow, presumably, the latest views. As research progressed, they might become five-guinea members in order to get the literature. But those herds might not attempt to do any lung examinations, because pigs would not be sold for breeding. The main purpose of the Association was really to give a stamp to people who wanted to say that their herds were free of virus pneumonia in order to set up other herds. In time, therefore, the Association might become more like a pedigree breeders' association and it would cater for the rather higher class pedigree breeder who wanted to sell proven stock.

They had no evidence to show that bacterial pneumonia had increased.

The Chairman asked if there was any suggestion that the number per square foot was being increased.

Dr. Goodwin replied that the pigs were not being intensely packed like broilers. The fattening house was defined as any enclosed building in which the pigs all breathed common air. Most people already had fattening houses of this type and they did not have to put up a special building. They were already there, in the ordinary mechanics of pig farming.

Mr. T. F. Vallely (Wolverhampton) asked whether anterior pneumonia was ever caused by conditions other than enzootic pneumonia, notably inclusion-body rhinitis, and, if it was, were the lesions indistinguishable from enzootic pneumonia?

Dr. Whittlestone said that it was almost impossible to answer Mr. Vallely's question simply. It had been reported from Sweden that experimentally-induced inclusion-body rhinitis had been associated with an anterior lobe pneumonia which was histologically indistinguishable from enzootic pneumonia. These workers thought that it was possible that enzootic pneumonia and inclusion-body-rhinitis were one and the same condition, but there was really not enough information to discuss the subject sensibly at the moment. Whether the two conditions were related to each other, or whether they just happened to occur together, was not known.

The whole question of the relationship of inclusion-body-rhinitis to enzootic pneumonia was in the melting-pot.

Mr. Vallely said that a herd in his area appeared to have inclusion body rhinitis which gave cause for worry. At one stage this herd was affected with enzootic pneumonia but this had now been eradicated. This suggested that two different entities were involved.

Dr. Whittlestone said that it did not mean that they were different things. He also had evidence that inclusion-body-rhinitis could be detected in herds not affected with enzootic pneumonia, as they understood it. In Sweden, however, it might be said that enzootic pneumonia was present in these circumstances.

A Member asked if it would be possible to give some idea of the incidence of the disease when doing a preliminary survey. What proportion of herds finished up by becoming virus free?

Dr. Goodwin replied that if a preliminary survey were made of a herd and 40 lungs examined (as a representative sample from the bacon factory) and the incidence of affected lungs was less than 5 per cent., there was a high chance that that would be one of the herds in the country that happened to be free from virus pneumonia. In other words, the 5 per cent. level was a very safe level. In most herds with virus pneumonia, the incidence of pneumonia was running at well above the 5 per cent. level. The latter level had been set because one could not always have a herd without any pneumonias; occasionally a pig would inhale straw and get a foreign-body pneumonia, for example. Somewhere, there was a dividing line. They were probably paying a price for putting it at 5 per cent., because they were catching all sorts of other respiratory fish in their net; but in a few years' time, experience might allow a shift up to, say, 15 per cent.

Mr. Smith said that most of the herds investigated were probably considering eradication; the percentage of pneumonias in the herds seen by the authors, therefore, was probably either very high or low.

Dr. Goodwin said that the man who came to one for

advice was usually worried because his pigs were coughing and growing badly. As a result, they themselves would indeed tend to have more experience of those herds.

Mr. P. L. Shanks (Aberdeen) thought that allowing 5 per cent. incidence was running a considerable risk. In the northern area of Scotland, he could think of at least two farms which had less than 5 per cent. pneumonia. One farm was well run and young pigs were kept for eight weeks by themselves, and then put in the fattening house. He was satisfied that there was enzootic pneumonia on that farm, but he would not recommend them to sell pigs as free from pneumonia. The pigs were in the same building but completely isolated, in litters.

Dr. Goodwin said that the regulations ruled that all the pigs had to breathe common air. The example just given would be of a non-fattening-house herd as far as the regulations were concerned. If a farmer wished to comply under the non-fattening-house regulations, every litter on the farm was considered as an individual unit. Every single litter had to have its lungs examined and the regulations were also much more severe. It might be possible, of course, to reduce the incidence of pneumonia to less than 5 per cent. if the pigs were spread out and isolated, but then a completely different set of regulations applied, where no pneumonias were allowed at all without further investigation, because the right conditions for the build-up of disease were not being created.

There were quite a number of respiratory diseases known in the Association that were believed to be different from enzootic pneumonia and they were not going to try to control them until something was known about them. All they were trying to do now was control a serious persisting pneumonia of the bacon pig that had a great economic effect on a herd basis. There were a number of pneumonias in little pigs that resolved before the pigs reached the bacon house, so that they did not appear as pneumonias when the pigs were slaughtered. These pneumonias got through the regulations. The more they had of that type, the happier they would be, because their net was not designed to catch that type of disease. They existed, however, and they were well aware of them.

It was interesting to see the effect of weather on these respiratory diseases. Just before coming to the Congress, he had been very busy after the weather had turned damp examining pigs that were coughing in herds in the Association. Probably most of these outbreaks would turn out to be respiratory diseases other than enzootic pneumonia. They usually came and went in a herd, although they sometimes hung on in successive litters of little pigs for years.

Dr. Whittlestone said that the whole crux of the matter was how enzootic pneumonia was defined, and the Association's concept of it was different from the non-persisting pneumonia described by Mr. Shanks. If enzootic pneumonia were present, one feature of it was that it would persist in a fattening house that was constantly being supplied with young pigs. Pneumonias that behaved differently were judged to be different diseases. Perhaps that approach was wrong, it remained to be seen. The disease described by Mr. Shanks would appear to be rather similar superficially to some mentioned in the paper. These were principally pneumonias of young pigs that had often largely resolved by bacon weight.

Dr. Watt (Edinburgh) referred to the use of the word "most" together with percentages—that at 5 per cent. it was hoped that most of the herds infected with virus pneumonia would be caught. Could a veterinary surgeon with utmost confidence tell a client to buy-in pigs from herds on the Association's list if his own herd was free from pneumonia; and if not, what reservations had to be made? That was a question he had been asked by people with pneumonia-free herds wishing to introduce new blood.

Dr. Goodwin replied that he did not remember saying that the 5 per cent. regulation would not catch enzootic pneumonia. At the moment, there was no evidence that it did not catch the disease; that was the whole basis of the thing, that enzootic pneumonia could not exist, under intensive conditions of management, below the 5 per cent. level. If that were untrue, some radical alteration had got to be made, or alternatively, the 5 per cent. level had to be pushed lower. On the question of how much reliance could be placed by a prospective buyer on the list published by the Association, that had been dealt with in the paper. To summarise, it had first of all to be assumed that their assumptions about enzootic pneumonia were true, which might not be so. In other words, there might occasionally be a herd that had enzootic pneumonia that would not be picked up by the regulations. They were not so dogmatic that they thought it could not happen but, so far, there was no evidence that it had happened. It was possible, for example, that there might be a vector they did not know anything about; it was also possible that enzootic pneumonia could exist in an attenuated form, but for all practical purposes it was thought that the regulations detected the disease.

The second known weakness was that one was always checking behind the progress of the disease. Thus the disease might be occurring for the first time in the little pigs, but it would be some while before it became apparent in the veterinary surgeon's certificate at the bacon factory end. There was always a time lag. Everyone was conscious of that, and therefore, most cautious people put their newly-bought pigs in isolation until such time as the herd from which they came got one more clear certificate. There was also an administrative lag in the publication of the Association's lists. They would come out every six months, but only after the veterinary surgeon's certificates had been received and checked.

Lastly, any control scheme could only be as sound as the integrity, conscientiousness and ability of the practitioner who did the signing of the certificates.

Dr. J. T. Stamp (Moredun, Edinburgh) said that he was confused by the 5 per cent. level used by Dr. Goodwin, who had said that the herds dealt with were the ones which were badly affected and that people only came to Dr. Goodwin because the herd was in a bad state. What evidence was there that there were not a considerable number of herds in the country where the farmer was not worrying at all since the incidence of pneumonia in such herds was, say, only 10 per cent.? If this was accepted then there was no reason why a herd should not be affected at the 5 per cent. level and hence the disease would be missed. He thought it was perhaps rather dangerous to argue that a disease must always be rampant especially after selection procedures have been carried out. With most diseases the herd incidence rate can vary from 1 per cent. to 100 per cent.

Dr. Whittlestone replied that he agreed that there were enormous biological variations. The pool of pigs being sampled for pneumonia was continually being replenished with young stock; the pool was never empty. In their paper they said that they had had to make a number of assumptions and they had made them in the light of previous experience with enzootic pneumonia. They had made what seemed to him to be a very reasonable assumption, in view of the fact that in all the herds studied where enzootic pneumonia was present, there had been a high incidence of pneumonia in bacon pigs from intensive fattening houses. A 5 per cent. incidence of consolidated lesions was tolerated to save the farmer unnecessary expenditure on histological examinations of occasional lesions. That was one of the assumptions that would be tested as the scheme progressed. It was a matter on which people could hold different opinions. If enzootic pneumonia could exist in a herd over a period of 18 months and more, without the incidence of consolidated lesions in lungs from pigs from the fattening house exceeding 5 per cent., then the scheme was unworkable as it stood.

Dr. Goodwin said that 60 certificates from veterinary surgeons had been received in the last six months, so that there were about 60 herds in the country that had reached the stage of submitting veterinary certificates. The veterinary problem might grow if there were a few hundreds or thousands, but so far the scheme seemed to be holding up. They were not worried yet that the disease was persisting undetected; there was no epidemiological evidence for this. Some breeders who had enzootic pneumonia badly in their pigs went in for periodic de-population; they cleared all pigs out of the fattening houses and then moved a new population in. That was how some people who had enzootic pneumonia badly tried to live with it. But the

(*Concluded at foot of col. 1 overleaf*)

Clinical Note

A Case of Anthrax in a Cat and in a Monkey

J. H. H. Cripps and R. C. Young
Shaftesbury, Dorset

WE thought this case might be worth recording as there appears to be very little reference, in the literature, to anthrax in the cat. This animal, so often being fed on raw knacker meat, must frequently be exposed to the risk of infection until knackeries come under veterinary inspection.

A 4-year-old Siamese cat was brought to the surgery for *post-mortem* examination. This cat was in good condition and appeared to be normal at 7.30 a.m. but was found dead at 2.30 p.m. The outward appearance was normal, with no signs of discharge from the mouth or the rectum. Internally, except for considerable congestion in the lungs, there appeared to be no abnormality. The blood was dark and unclotted, but as the cat had not long been dead, no significance was attached to this at the time. The spleen was not enlarged. A blood smear was taken and stained with methylene blue; it was found to be teeming with organisms morphologically indistinguishable from *Bacillus anthracis*, though capsules were unconvincing and there was no McFadyean reaction.

When the owner was informed of the diagnosis, he told us that his pet 3-year-old Mona monkey had just been found dead, and that once again there were no previous signs of illness. It transpired that the cat had been fed on raw knacker meat and that the monkey had been given a small piece of this.

A blood smear was taken from the monkey and stained with methylene blue; it showed typical *Bacillus anthracis*, with capsular staining and a typical McFadyean reaction.

It appears from the observations above that there is no very striking macroscopic *post-mortem* finding in a cat that has died from anthrax. Perhaps for this reason anthrax in the cat may occur rather more frequently than has been diagnosed.

Scheme for Supervising Pigs.—*Concluded.*

Association's regulations tried to force the disease the other way, by saying that the fattening house must never be emptied; it must be continually fed with susceptible pigs until there had been 18 months of clear lung examinations. Only then was the herd admitted to the list for the first time.

The Chairman said that the session must not be concluded without a hearty vote of thanks being accorded to the two speakers. The subject was one which was close to his heart and he had, over the last two years, made great efforts to find out what Drs. Goodwin and Whittlestone were up to. He had been tremendously impressed by their success, their painstaking work, and the absolute conscientiousness with which the programme had been carried on. It had been of the utmost importance for practising veterinary surgeons who were interested, and who had clients who were interested in the scheme, to come to grips with the two speakers. Veterinary surgeons had to know intimately the people in charge of the scheme, and each had to make up his own mind on it. He personally had no doubt whatsoever that the scheme was in the best possible hands that it could be in, in Britain to-day.

The vote of thanks was carried by acclamation.

Clinical Note

Acute Allergic Response to Penicillin

P. B. Clarke
New Town, Uckfield

THE following case is recorded as a matter of interest on a subject which has been very neglected in the veterinary field.

Subject. A 2-year-old pedigree Beagle bitch just weaned from her first litter of 8 puppies.

History. After whelping on May 2nd, 1960, the bitch had been fed on 2 pints daily of a pasteurised bulk milk sample. The owner noticed a copious discharge of haemorrhagic mucus from the vulva on May 11th, but this had apparently improved after a single dose of 300,000 units procaine penicillin G intramuscularly. This was the first time the animal had ever received penicillin treatment.

Clinical examination. On July 6th, 1960, the bitch was reported to be eating well. She had lost some condition during her lactation period but appeared quite healthy except for a recurrence of the vulval discharge.

Diagnosis. A low-grade infection of the genital tract.

Treatment. A pessary* containing dibromopropamidine and streptomycin was inserted into the anterior fornix of the vagina. The bitch was injected with 300,000 units of procaine penicillin G intramuscularly. Fifteen minutes later the bitch began to shiver, and then salivate. Discomfort became progressive. Vomiting occurred 3 times. In the meantime the abdomen became very red, and urticarial blebs appeared on the skin which was obviously very irritating; the bitch laid down on cold surfaces between panting, scratching and restless wanderings. No further treatment was given and the acute symptoms gradually subsided in a matter of hours.

Discussion. It is suggested that this is a clear case of an acute allergic reaction to treatment with penicillin. In view of the rarity of such reactions (despite the widespread use of penicillin), and the fact that the bitch had receved only the one previous penicillin treatment, it is possible that the sensitising doses of penicillin may have originated in the copious quantities of milk consumed.

* Solcon: May and Baker Ltd.

THE ROYAL COLLEGE OF VETERINARY SURGEONS

Library

The packing of the library for removal will commence on Thursday, December 1st, 1960, and the actual removal will be carried out over the week-end commencing Friday, December 16th.

From Thursday, December 1st onwards, until further notice, it will be possible only to provide a limited library service.

As soon as the library in the temporary College building is available to give full services a notice will appear in THE VETERINARY RECORD.

Diagnosis of Rinderpest by Lymph Node Biopsy

BY

R. D. BROWN and G. R. SCOTT

East African Veterinary Research Organisation,
Muguga, Kikuyu, Kenya

SUMMARY.—Biopsy samples of lymph nodes of cattle infected with rinderpest were tested for diffusible rinderpest antigens by the agar gel double-diffusion precipitation technique. Positive samples were obtained on the first to the eighth day after the start of the fever. The highest percentages of positives occurred in samples taken on the third to the fifth day. The implications of the technique for confirming rinderpest in the field are discussed.

Introduction

IN years past rinderpest was diagnosed readily on the history and the clinical and *post-mortem* findings. Laboratory confirmation was seldom required. When necessary, tissue suspensions from suspect animals were inoculated into known-susceptible and known-immune cattle. In East Africa mass vaccination with caprinised rinderpest virus has altered the epizootiological picture in the remaining enzootic corners such that oubreaks are confined largely to weaners. Most adults have been actively immunised and sucklings are protected through the passive transfer of antibodies in the colostrum. Recognition of the disease is difficult and confirmation of a provisional diagnosis is usually required. A further complication stems from the recent recognition of several rinderpest-like diseases such as bovine rhinotracheitis and virus diarrhoea in North America and so-called mucosal diseases in America, Europe and Australia.

White (1958) made diagnosis of rinderpest easier by the demonstration of a diffusible rinderpest antigen by the agar gel double-diffusion precipitation technique. He preferred antigens prepared from mesenteric lymph nodes harvested from cattle killed on the fifth day after the onset of fever. Nevertheless he did detect the precipitinogen in mesenteric lymph nodes collected as early as the second day and as late as the seventh day. Stock-owners are reluctant to sacrifice animals in the early acute phase of the disease for diagnostic purposes. They prefer that moribund or dead animals be so used. Unfortunately the mean day of death of rinderpest-infected cattle is 7 to 8 days after the start of fever, consequently a considerable number of animals die after this period (MacOwan, 1956).

The distribution of rinderpest diffusible precipitinogen in infected cattle has been determined (Scott & Brown, 1960). All carcase lymph nodes contained the antigen and the logical extension of this finding to the diagnosis of the disease in living animals by testing biopsy samples is herein recorded.

Materials and Methods

Cattle

The cattle were crosses between European breeds and East African Shorthorn Zebus, the former predominating. They were about 2 years old and weighed 150 to 300 kg. All were reacting to Kabete "O" rinderpest virus administered parenterally.

Biopsy Technique

Lymph node samples were obtained from the right or left prescapular lymph nodes of cattle on various occasions during the 9 days which followed the start of the fever. Hypodermic needles of 30 mm. length and 14 gauge were attached to a 5-ml. or 10-ml. syringe. The needle was pushed through the skin into the substance of the lymph node. The plunger of the syringe was withdrawn a few times and varying amounts of tissue passed into the needle and syringe. Usually one or two lymph node penetrations sufficed to obtain enough material to fill one or two cups in an agar plate. Most cattle were sampled twice but a few were sampled three times. A total of 20 biopsies were done on each of the 9 days which followed the start of the fever.

Agar Gel Diffusion Test for Precipitation Antigen

The biopsy samples were tested by the agar-gel diffusion test for rinderpest antigen described by White (1958). The samples were diffused against rinderpest hyperimmune sera prepared in rabbits. A positive rinderpest antigen, consisting of a 33⅓ per cent. suspension of lymph nodes taken from rinderpest-infected cattle on the 4th day of pyrexia, was included in each test. Biopsy material, positive antigen and hyperimmune serum were placed in cups 4 mm. in diameter placed 4 mm. apart in the agar plates. The plates were examined 24 hours later for lines of precipitation. Biopsy samples were considered positive if precipitation lines developed which were confluent with the reaction line between the positive antigen and the hyperimmune serum.

Results

TABLE I

RESULTS OF TESTING LYMPH NODE BIOPSY SAMPLES FOR RINDERPEST PRECIPITATING ANTIGEN

Time of Biopsy after start of Fever (days)	Number of Positive Biopsies	Number of Negative Biopsies	Percentage Positive
1	1	19	5
2	6	14	30
3	10	10	50
4	17	3	85
5	11	9	55
6	4	16	20
7	3	17	15
8	1	19	5
9	0	20	0

Tests of 180 lymph node biopsies from 81 animals were made (Table I). The distribution of positive biopsies taken on the 1st to the 9th days after the onset of fever is shown in Fig. 1. Positives occurred

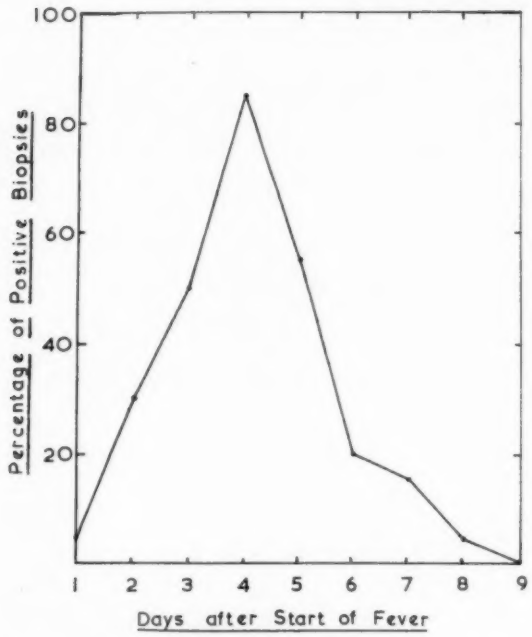

FIG. 1.—Distribution of Positive Biopsies in Rinderpest-Infected Cattle.

on the first to the eighth day inclusive. The greatest number of cattle were positive when tested on the 3rd, 4th and 5th days.

The only lines of precipitation obtained when the biopsy materials were tested were all confluent with those obtained with the control antigen; in other words non-specific reactions did not occur in this series.

Discussion

Our findings indicated that rinderpest could be diagnosed in reacting cattle by testing lymph node samples removed by needle biopsy. The optimal period for successful diagnosis was the 3rd to 5th day of fever when 63.3 per cent. were positive. Animals in the very early and late stages of a rinderpest reaction were less likely to give positive results. Of the 120 biopsies made on the 1st and 2nd and 6th to 9th days only 15 (12.5 per cent.) produced lines of precipitation against hyperimmune serum.

The technique and test are simple and could be used in the field to confirm rinderpest provided that as many sick animals as possible are tested. The overall probability of a positive occurring in our sampling period was 1 in 3.4 times. In other words at least 4 animals would have had to be sampled before a positive could be expected.

Diarrhoea is a prominent sign of rinderpest. The onset occurs in infected cattle around the third to the fifth day of fever. Such animals would be expected to yield a positive biopsy sample but cattle which have had diarrhoea for several days and show dehydration and emaciation would probably give negative results.

Rinderpest can be diagnosed by testing lymph node biopsy material for complement fixing antigen (Brown & Cowan, 1960) but the technique for such a test is more complex than the agar gel diffusion test.

Acknowledgment.—This paper is published by permission of the Director of the East African Veterinary Research Organisation.

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Efficiency in Livestock Feeding

In view of the cordial relationship which exists between the B.V.A. and the animal feeding-stuffs industry we have been asked to publish the following joint statement issued by the National Agricultural Advisory Service, the Compound Animal Feeding Stuffs Manufacturers' National Association, the National Association of Corn and Agricultural Merchants, the National Association of Provender Millers, and the National Council of Concentrate Manufacturers.

During the past year, discussions have taken place between the N.A.A.S. and trade associations representing the feeding-stuffs industry. These have centred on the feeding of farm livestock, and, in particular, on the approach to giving information and advice to the farmer as to feeding methods and policies. Both parties agree that in giving such advice it is essential to take into account those factors capable of influencing the result of adopting alternative feeding methods on the farm concerned.

This memorandum outlines some of these factors and presents an objective viewpoint on them as a basis for advice.

1. *National Food Supplies for Livestock Production*

Nationally we have available from our own farms, grass and conserved grass products, root crops, cereals, etc., but it is a fact that notwithstanding all known scientific advances it is only with the aid of about 5 million tons of imported foods per annum that we are able to produce the present proportion of our human food requirements.

There is scope for increasing livestock production, by producing more stock foods on our own farms and by increasing the efficiency with which we convert animal food into livestock production.

2. *More Livestock Foods off the Farm*

Every farm should make its maximum possible contribution towards supplying the total foods required to achieve optimum profitable production on that farm. This applies especially to ruminants which convert grass into milk and meat, but production of grass and of conserved grass products is only useful if these are converted efficiently into milk and meat. The quality of farm-grown feeds is just as important as their quantity and there is room for more effort to improve and, where practicable, to assess this quality.

Wasteful feeding can occur through incorrect evaluation of farm-grown foods, just as it can through unnecessary or inaccurate feeding of purchased foods, either through failure to convert foods efficiently, or through failure, by underfeeding, to achieve potential production levels. Waste can also occur through the use of unsuitable farm feeding equipment.

3. *Variations in Farm Circumstances*

No two farms are alike, and generalisations are dangerous and rarely help the farmer. Each farm has to determine its policy, taking into account many aspects, including land and soil, buildings, equipment, labour and housing, available capital, standard of management, and genetic quality of stock kept. Optimum levels of production, and the best use of home-grown foods, can very rarely be achieved with no use of purchased feeding-stuffs, but the extent of such purchases and their form will vary from farm to farm, and even between different classes of stock on the same farm. Every farmer should be made aware of the very wide range of foods available to enable him to get the best out of his farm and his stock.

4. *Flexibility of Purchase in Feeding-stuffs*

Private and independent research has led to a better understanding of animal requirements and the formulation of a wide range of feeding-stuffs based on scientific knowledge. Farmers have available to them straight feeding-stuffs ingredients, and a wide choice of compounds and concentrates. The N.A.A.S. are equipped to give guidance and advice on the feeding of livestock, taking into consideration the economic factors of the individual farm.

Feeding-stuffs manufacturers offer a wide range of products of which the broad classes are:—

(*a*) Pre-mixed protein-mineral-vitamin concentrates or balancers, specifically formulated so that when mixed at recommended rates with cereals or other straight ingredients, a balanced diet is produced for the class of stock concerned.

(*b*) Complete compounds.

Thus, whether a farmer buys compounds or mixes on his farm, he has the fullest access in either case to the benefits of modern nutritional knowledge embodied in the one case in the compounds he purchases, and, in the other, in the protein-mineral-vitamin concentrates available to him for mixing with his cereals.

Farmers have open to them, therefore, complete flexibility between a "mix-all" and a "mix-nothing" policy for feeding each class of their stock. Used on an informed basis these alternatives fulfil the needs of the three main categories of farms in the country, which can be arbitrarily summarised as:—

(i) Holdings, especially small dairy farms and intensive pig and poultry farms, with limited land, where the farmer relies on purchased feeding-stuffs to enable him to show reasonable profits.

(ii) Large-acreage holdings where it is possible to meet a large part of the nutritional requirement from the farm itself, use being made of balancers and concentrates, or purchased proteins, etc., to rectify the balance of the diet.

(iii) A very large group of intermediate farms, on which it is open to the farmer to decide on the relative profitability of the different alternatives.

5. *Assessment of Costs of Livestock Products*

Both the N.A.A.S. and the trade associations of the feeding-stuffs industry are agreed that the price per ton of livestock feeds is not the only yardstick for comparisons or advice. The measure of efficiency and profitability for the farmer is the cost of the feed in relation to the production which follows its feeding. Farmers can establish this cost and so adopt feeding policies on an informed basis, only by the use of recording systems, and a much wider use of these is recognised as highly desirable, whilst the actual form of records deserves further study to make them simple to keep, yet of maximum use as management guides.

At the same time, since costs are bound to be calculated to arrive at such comparisons, it is essential that all figures are fully realistic, and take into account all costs incurred up to the time of feeding to the animal. It is suggested that the following factors must be accounted for:—

(*a*) The relative cost of capital and operating charges including labour, power, repairs, insurance, depreciation, interest on capital, additional storage according to the system of production and the deterioration of raw materials.

(*b*) The whereabouts of the materials, *i.e.*, whether they have been produced on the farm or expense has to be incurred in transporting them there.

(*c*) The scale on which the individual ingredients can be purchased, *i.e.*, whether they can be obtained at reasonable prices delivered direct from source of whether it is necessary to buy in small quantities involving extra charges for storage and re-delivery by the local merchant.

(*d*) The degree of complexity of the most suitable formula. Simple formulae based on complete concentrates or balancers may be more easily and accurately mixed on the farm than elaborate mixtures containing several ingredients required in small proportions.

(*Concluded at foot of col. 2 overleaf*)

News and Comment

THE FOOT-AND-MOUTH OUTBREAK

Listed elsewhere are the addresses, supplied by the Ministry, where foot-and-mouth disease is occurring; these are up to date as at the first post on Wednesday morning, our closing time for press. At that time the outbreak—the worst for a number of years—was still spreading despite all the exertions of veterinary surgeons in the Animal Health Division, and the latest Ministry report announced that new controlled areas were Bedford, Bucks., Cambridge, Derbyshire, Hertfordshire, Huntingdonshire, Isle of Ely, Leicestershire, Lincs. (Holland), Lincs. (Kesteven), Lincs. (Lindsey), Middlesex, Northants., Notts., Oxfordshire, Rutland, Soke of Peterborough, Staffs., Warwickshire, Worcestershire, the West Riding of Yorkshire, the administrative County of London, East Ham, West Ham, and the administrative County of Essex.

A total of 110 outbreaks have been reported since the epidemic began on November 6th, and some 18,000 animals have been slaughtered. Strict watch is being kept at the Irish ports against importation of possibly infected animals, and it is reported that several veterinary surgeons from Northern Ireland have gone to the help of their colleagues in Scotland.

The source of the outbreak has not yet been established with certainty, though it is stated that Ministry veterinary officers suspect infected pig swill from an imported carcase in the Newcastle area. Questioned in the House, the Minister of Agriculture announced that from February, 1961, the importation of pork from Argentine, Chile, Uruguay and Brazil would be prohibited. The reason for the delay was the notice which had to be given to these countries. There have been rumours that the National Farmers' Union were pressing the Government to forbid the importation of beef from these countries, but a Union spokesman has informed us that these rumours are without foundation.

Our readers will not need to have described to them the herculean efforts of their colleagues in the Animal Health Division, which are deserving of the highest admiration and praise. Should the situation worsen the Division might be glad to have a list of private practitioners on whose temporary help they could call. Large private practices especially, who could spare, at a pinch, a partner or an assistant are asked to get into touch with Mr. J. C. Wallace at the Ministry of Agriculture, Hook Rise, Tolworth, Surbiton, Surrey (Telephone: Derwent 6611) so that a register of names can be compiled.

WORLD VETERINARY POULTRY ASSOCIATION CONFERENCE

The first conference of this Association was held in Utrecht on November 14th, 15th and 16th. The programme of papers included the following: *A Proposed Classification of the Leucosis Complex and Fowl Paralysis* by Dr. J. G. Campbell; *Haemorrhagic Syndrome* by Dr. H. Marthedal; *Duck Plague* by Professor J. Jansen; *Avian Encephalomyelitis* by Dr. Guillon; *Prophylactic Effect of vitamin A on Caecal Coccidiosis* by Professor Gerriets; *Hygiene of Poultry Husbandry and Feeding* by Dr. Siegmann. There were upwards of 200 participants from some 18 countries, and the conference was held in the hall of the Veterinary Institute of Anatomy. Among the delegates from Great Britain were the following: Dr. R. F. Gordon, secretary of the World Veterinary Poultry Association, P. K. Ashton, R. A. A. Beament, W. E. Beattie, J. D. Blaxland, Dr. W. P. Blount, Dr. P. M. Biggs, Dr. H. P. Chu, R. H. Duff, M. H. Fussell, Dr. J. S. Garside, L. A. Hemsley, Dr. D. G. Howell, S. A. Keeble, Mrs. K. M. Lowndes, J. M. S. Lucas, Dr. W. M. McKay, R. I. Macrae, J. Reid, W. N. Scott, Col. V. Short, T. E. Singleton, K. H. B. Wood and Dr. W. R. Wooldridge.

NEW VETERINARY RESEARCH UNIT

Smith Kline & French Laboratories Ltd., announce that they have purchased Tallents Farm, Kimpton, Hertfordshire, which will be developed as a veterinary research unit. It will provide facilities for the evaluation, under field conditions, of the Company's products.

HIGH POTENCY INJECTABLE IRON AND GROWTH RESPONSE IN PIGLETS

Recent trials carried out in this country by Anne Walker, under the direction of Dr. John Taylor, of Cyanamid, have been designed to show the significance of the use of high potency injectable iron on the growth response of piglets. The findings were revealed at a press conference last Monday afternoon when it was made known that the full data would be published fairly shortly. Meanwhile, the new iron solution, containing 100 mg. of iron per ml. is available commercially.

The product is said to demonstrate a notable advance in the use of injectable iron for the prevention of anaemia in piglets reared indoors. Information gained from the 60 litter trial showed that piglets gain 6 per cent. more bodyweight at weaning on 200 mg. of iron compared with the 150 mg. at present in general use. The increase in weight did not bring about a decrease in the level of haemoglobin in the blood, despite the fact that the heavier piglets would be expected to have a larger volume of blood.

Efficiency in Livestock Feeding.—*Concluded.*

(*e*) The efficiency and thoroughness of farm-mixing compared with factory standards.

(*f*) The form in which the ration can be handled and fed most economically, *i.e.*, meals, pellets or nuts.

6. *Influence of other Factors than Feeding*

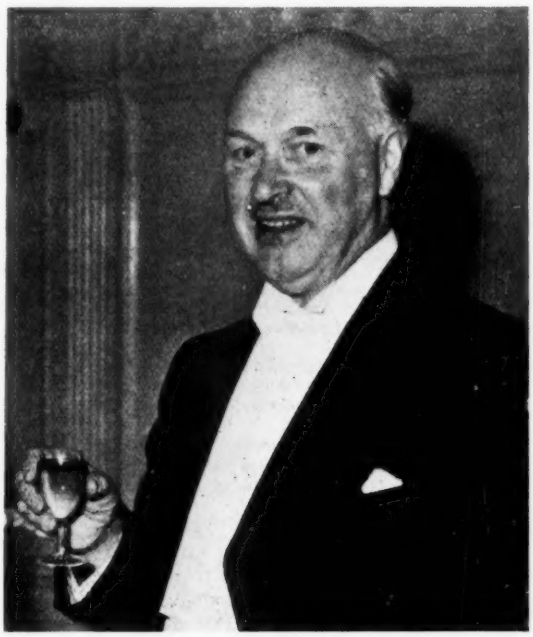
Whatever the form of food used, the success and efficiency of any livestock project depends not only on feeding but on breeding and management as well. The results of feeding the food will be influenced by such factors as the genetic potential of the stock, their health and hygiene and the level of stockmanship.

RETIREMENT OF MR. F. KNIGHT

Readers of THE VETERINARY RECORD will recall that tributes, and a presentation, were made to Mr. F. Knight, the retiring General Secretary of the B.V.A. on the final day of the Glasgow congress. That date was chosen by the Officers as it was the last time Mr. Knight would be on duty at a Council meeting; but the actual day of his official retirement from our service was last Friday, November 11th, when he completed 35 years' service.

He is, indeed, part of B.V.A. history; for, in a way few others have done—and none so closely—he has seen the Association grow from a small, comparatively impoverished body having its offices in a couple of rooms, to a professional organisation of international status.

Mr. Knight is too modest to tell anyone, or even to ask himself how much of that remarkable development has been due to his own efficient, conscientious work. The answer is that he has laid the B.V.A. deeply in his debt. Its honorary officials over many years, would be the first to acknowledge the services of the permanent staff; and the quality of that staff has derived from its head. Mr. Knight has been that head for the best part of his working lifetime; and he leaves behind him an office machine of which any executive might be proud.



To his colleagues on the staff, this parting must be an occasion for sadness. He has been one of those who believed in pushing the cart from behind and not conspicuously pulling it from the front; in a word he has been modest and self effacing almost to a fault; but his influence has permeated every corner of our work and will long remain.

His senior colleagues, in particular, will miss his quiet kindliness, his singular talent for putting oil in the machine without appearing to do so; they will miss his profound knowledge of the Association's tradition, its precedents and its policy; perhaps most of all they will miss his prudence and downright north country common sense. All his staff, senior and junior, salute him, and wish him and his wife many years of good health in happy retirement.

On the day of his departure he was entertained to luncheon by the President, accompanied by Mr. John Anderson, the new General Secretary.

REGIONAL APPOINTMENTS IN THE N.A.A.S.

Mr. W. S. Gibson, B.SC. (AGRIC.), Regional Director of the National Agricultural Advisory Service, East Midland Region since 1951, will be retiring from the public service on November 30th, 1960. He will be succeeded by Mr. R. B. Ferro, N.D.A., at present Assistant to the Senior Advisory Officer (Agriculture) in London.

THE WORLD VETERINARY ASSOCIATION

The latest booklet of news items circulated by the W.V.A. says it is expected that a formal application for associate membership will be submitted to the Permanent Committee in May next year by the World Association of Small Animal Specialists. It is understood that the British Small Animal Veterinary Association proposes to invite to its 1961 Congress delegates from countries which support the World Association of Small Animal Specialists, and it will then be possible to discuss matters of common interest.

The year 1963, the 100th Anniversary of the initiation of the International Veterinary Congresses, is to be declared World Animal Health Year by the World Veterinary Association.

UNIVERSITY NEWS

Liverpool

Degree of B.V.Sc. Second Examination

PART III: ANIMAL MANAGEMENT

Bell, J. C.

PERSONAL

Mr. W. N. Scott, has recently been appointed Manager, Veterinary Development, to Smith Kline & French Laboratories Ltd. He joined the Overseas Veterinary Service in August, 1951, was appointed Assistant Director of Veterinary Services, Uganda in 1954, and Deputy Director, East African Trypanosomiasis Research Organisation in 1957. He retired from the Overseas Veterinary Service in June, 1960.

Dr. A. B. Paterson, who is in charge of the Virology Department of the Ministry's laboratory, Weybridge, is attending the East African Veterinary Research Organisation's laboratory at Muguga to take part in a course on rapid laboratory techniques for the diagnosis of rinderpest, mucosal disease and other similar conditions. He will also visit the Kenya Veterinary Research Laboratory at Kabete, and the Research Station of Messrs. Burroughs Wellcome and Company also at Kabete, and probably also, if time allows, the Virus Research Institute at Entebbe. Following this Dr. Paterson will go to the Veterinary

Laboratories at Onderstepoort to consult with fellow workers there on African horse sickness and other virus matters.

Mr. E. C. Hulse, the head of the laboratory's Biological Products and Standards Department, is visiting Turkey and Iran to look at the progress made in these countries following the Seminar on Anaerobic Disease which was held at Weybridge about a year ago.

Births

CRAIG.—On November 13th, 1960, to Winnie (*née* Galloway), wife of Gordon R. Craig, B.V.M.S., M.R.C.V.S., Beechwood, Galston, Ayrshire, a daughter, Alison Phillips.

JONES.—On November 8th, 1960, to Shirley (*née* Pearce), wife of Bruce V. Jones, M.R.C.V.S., of The Oaks, Clavering, Essex, a daughter, Sheridan Clare, sister to Julian.

MOODY.—On November 6th, 1960, to Caroline, wife of Peter W. Moody, B.V.M.S., M.R.C.V.S., Stone, Staffs., a son, Timothy Charles Warner.

STEELE.—On October 15th, 1960, to Patricia (*née* Parry), wife of Colin Steele, B.V.SC., M.R.C.V.S., 65 Seaview Road, Wallasey, Cheshire, a son, Robert John, brother for Catherine, David, and Christopher.

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

GOVAERTS, Louis Victor, 31 Farley Hill, Luton, Beds. Graduated December 22nd, 1953 at the Royal Veterinary College, London. Died November 6th, 1960, aged 33 years.

COMING EVENTS

November

19th (Sat.). B.S.A.V.A. SOUTHERN COUNTIES REGION. Meeting at the Royal Hotel, Winchester, 7.30 p.m.

23rd (Wed.). WESTERN COUNTIES VETERINARY ASSOCIATION. Annual Dinner Dance at the Imperial Hotel, Exeter, 7 p.m.

23rd and 24th (Wed. and Thurs.). B.V.A. CONFERENCE ON HYPOMAGNESAEMIA. Meetings at the Victoria Halls, Southampton Row, London, W.C.1, commencing 10.30 a.m. each day.

LAKELAND VETERINARY ASSOCIATION. General Meeting at the Fleece Inn, Kendal, 6 p.m.

24th (Thurs.). ROYAL (DICK) VETERINARY COLLEGE RUGBY CLUB. Annual Ball in the Adam Rooms, George Street, Edinburgh, 9 p.m.

25th (Fri.). WESTERN COUNTIES VETERINARY ASSOCIATION. Dinner-Dance at the Rougemont Hotel, Exeter, 7 p.m.

NORTH WALES DIVISION. Annual General Meeting at the Royal Hotel, Llangollen, 2 p.m.

28th (Mon.). EAST YORKSHIRE CLINICAL CLUB. Meeting at the Talbot Hotel, Malton, 8. p.m.

30th (Wed.). A.V.T. & R.W. SCOTTISH REGIONAL GROUP. Meeting at the Royal (Dick) School of Veterinary Studies, 2.30 p.m.

December

1st (Thurs.). LAKELAND VETERINARY ASSOCIATION. Annual Dinner Dance at the Old England Hotel Bowness-on-Windermere, 7.30 p.m.

2nd (Fri.). NORTH OF ENGLAND VETERINARY MEDICAL ASSOCIATION. Annual Dinner-Dance at the White Swan Hotel, Alnwick.

ROYAL COUNTIES VETERINARY ASSOCIATION. Ordinary General Meeting at the Caversham Bridge Hotel, Reading, 2.30 p.m.

MIDLAND COUNTIES VETERINARY ASSOCIATION. Joint Meeting with the Shropshire Veterinary Club at the Oak Hotel, Shelton, Shrewsbury, 8 p.m.

8th (Thurs.). ESSEX VETERINARY SOCIETY. Meeting in the Judges Room at the Saracen's Head, Chelmsford, 7.30 p.m.

9th (Fri.). BRITISH VETERINARY POULTRY ASSOCIATION. 14th Meeting of the Association at Unilever House, London, 2.15 p.m.

14th (Wed.). SUSSEX VETERINARY SOCIETY. Meeting at the Old Ship Hotel, Brighton, 2.30 p.m.

TECHNICAL DEVELOPMENT COMMITTEE

The next meeting of the Technical Development Committee will be held at 7, Mansfield Street, London, W.1, at 10.30 a.m. on Monday, December 5th, 1960.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak

Anthrax

Berks. Rookwood Farm, Stockcross, Newbury (Nov. 11).
Sussex. Nut Brown Farm, Hooe, Battle (Nov. 8).

Foot-and-Mouth Disease

Aberdeen. Home Farm, Inverquhomery, Longside, Meikleton of Lessendrum, Drumlade; Braehad Farm, Cruden Bay; Avaulds Farm, Turriff; Gateside Farm, Udny (Nov. 10); Belmont Mart, Aberdeen; Balring Farm, Mintlaw Station; Westburn, Balmedie (Nov. 11); Woodhead, Cairness Fraserburgh; West Auguhadiae, Auchnagatt; The Slaughterhouse, Hutcheon Street; Shavado Home Farm, Bruchley Estates, Maud; Alton Farm, Keithhall, Inverurie; North Broadmuir, Cruden Bay; Hay Farm, Cruden Bay; Bing Hill Farm, Milltimber; Central Mart, Aberdeen (Nov. 12); Lapplan Brae, Old Deer (Nov. 13); Delliston, Auchnagatt; The Guise, Tough, Alford; Green Myre, Rattray, Peterhead (Nov. 14).
Cambs. East Fen Farm, Isleham, Ely (Nov. 8).
Clackmannanshire. Bogside Farm, by Alloa (Nov. 13).
Durham. Whitehouse Farm, Chester le Street (Nov. 9); 11, Front Street, Chilton Moor, Houghton-le-Spring (Nov. 10); Flinton Hill Farm, Sunderland (Nov. 11); The Abattoir, 49, North Bridge Street, Sunderland (Nov. 12); The Moors Farm, Winters Bank, Fencehouses, Houghton-le-Spring (Nov. 14).

Kincardineshire. Upper Craig Hill, Arbuthnott, Laurencekirk (Nov. 13).

Lanarks. Melbourne Street, Glasgow, C (Nov. 12); Dalmacoulter and Roughcraig Farms, Airdrie, Hamilton (Nov. 14).

Leics. Upper and Lower Burn Farm, Great Bowden (Nov. 10).

Norfolk. Lower Farm, East Tuddenham, Dereham; Bungalow Farm, Barford, Norwich; Moat Farm, Mattishall, Dereham; Mill Farm, Elsing, Dereham; West End Farm, Tibenham, Norwich; Church Farm, New Buckenham, Norwich; Manor Farm, Framingham Pigot, Norwich; Old Hall Farm, Mattishall, Dereham; North Bridge Pasture, Little Cressingham, Watton; Wood Rows Farm, Deopham, Wymondham (Nov. 8); Low Farm, South Walsham, Norwich; 7, Holding, Thurne, Great Yarmouth; Beech Farm, Bunwell, Norwich; Park Farm, Hevingham, Norwich; Dane Moor Farm, Hardingham, Norwich; Church Farm, Meadows, Southacre, Swaffham; Laurels Farm, Ludham, Norwich; Church Farm, Sidestrand, Cromer; Dudwick Farm, Buxton, Norwich (Nov. 9); Hoveton Old Hall, Neatishead, Norwich; Church Farm, Barford, Norwich; The Limes, Marston Lane, Norwich; Anchor Grove Farm, Brandon Bank, Downham Market; Street Farm, Brampton, Norwich; West Grove, Martham, Great Yarmouth; Holly Farm, Horningtoft, East Dereham (Nov. 10); Hall Farm, Hindringham, Fakenham; Manor Farm, Drayton, Norwich; The Dotsel, Edgefield, Melton Constable; Orchard View, Saxlingham, Norwich; Barton Farm, Sea Palling, Norwich; Flordon Hall Farm, Flordon, Norwich; Whinmere, Hickling, Norwich (Nov. 11); Sturgeon Farm, Roydon, Diss; Woodland Bungalow, Felthorpe, Norwich (Nov. 12); Meadow Farm, Cringleford, Norwich; Abbey Farm, Castle Acre, King's Lynn; Preston Green Farm, Tibbenham, Norwich; Lower Buildings Hall Farm, Mundham, Norwich; Hookwood Farm, Morley St. Peter, Wymondham (Nov. 13); Pentney Common, Pentney, King's Lynn; Haugh Farm, Barnham, Norwich; The Rum, Tottenhill, King's Lynn; Rookery Farm, Metfield Harleston (Nov. 14).

Northumberland. West End Farm, Berwick Hill, Ponteland, Newcastle upon Tyne; 1, Sootswood Road, Newcastle upon Tyne (Nov. 8); Dene Head, Wallboltle, Newcastle upon Tyne; Manor Slaughterhouse, Alnwick (Nov. 9); Howden Dene Piggery, Howden on Tyne; Smallburn Farm, Ponteland, Newcastle upon Tyne; Slaughterhouse, Lime Street, Newcastle upon Tyne (Nov. 10); New Houses, Hallington, Newcastle upon Tyne (Nov. 11); Horsley Hill Farm, Horsley on Tyne, Newcastle upon Tyne (Nov. 13).

Notts. Lodge Farm, Burton Joyce (Nov. 14).

Stirling. Netherinch, Milton at Campsie (Nov. 13); Easter Drumbroider, Avonbridge; Twechar Farm, Twechar (Nov. 14).

Suffolk. Lower Hill Farm, Great Bradley; Ravens Hall Farm, Great Bradley; Grove Farm, Ilketshall St. Lawrence, Beccles; West Street Farm, Walsham le Willows, Bury St. Edmunds (Nov. 8); Park Farm, Shadingfield, Beccles; Park Farm, Wyverstone, Stowmarket (Nov. 9).

Fowl Pest

Beds. Pitstone Grange, Smallholding, Cheddington Road, Pitstone, Leighton Buzzard (Nov. 14).

Cambs. 98, Westfield Road, Manea, March (Nov. 11).

Lancs. Holts House Farm, Pittsea, Oldham (Nov. 8).

Norfolk. Carr Farm, Besthorpe, Wymondham (Nov. 8); Ashwren, Browick Road, Wymondham; Station Road, Yaxham, East Dereham; The Bungalow, Carleton Rode, Norwich; 7, Church Row, Weybread, Diss (Nov. 9); Kimberley Hall, Kimberley, Wymondham (Nov. 10); Glad House, Poultry Farm, Sheringham; Mendham Priory, Harleston, Diss; 19, Greenways, Carlton Rode, Norwich; School House, Snetterton, Norwich (Nov. 11); Waveney House, Weybread, Diss; Reddenhall, Harleston; Goose Green Farm, Hall Road, Winfarthing, Diss; Broomhill Holding, Hackford Vale, Reepham, Norwich; Manor Farm, Tibbenham; Irstead Lodge, Irstead, Norwich; Willow Farm, Saddle Bow, King's Lynn; Luch-Bush, Reddenhall, Harleston; Winsford Hall, Stokesby, Great Yarmouth (Nov. 12); Church Farm, Tunstead, Norwich; Briar Farm, Harleston (Nov. 13); Sarn Bank Farm, Roughton, Norwich; Pinelands Poultry Farm, Horsford, Norwich; Brookside, Breckles, Attleborough; Drive Farm, Norwich Road, Dereham; Home Farm, Fressingfield, Diss (Nov. 14).

Salop. Park House, Shawbury, Shrewsbury (Nov. 10).

Somerset. West Pennard, Glastonbury (Nov. 8); Church Farm, Ash, Martock (Nov. 13); Bridge Farm, Coat, Martock (Nov. 14).

Suffolk. Klondyke Allotments, Bury St. Edmunds (Nov. 8); Brights Farm, Bramfield, Ipswich (Nov. 9); 44, Fornham Road, Bury St. Edmunds; Home Farm, Great Finborough (Nov. 11); Nostra Domus, Furze Close, Thurston, Bury St. Edmunds (Nov. 12); The Green, Stow Upland, Stowmarket; Homeleigh, Wayneford Road, Ditchingham, Bungay (Nov. 13).

Yorks. Battman House Farm, Nun Monkton (Nov. 10).

Swine Fever

Ches. King Street Hall Farm, King Street, Rudheath, Middlewich (Nov. 11); Blue Bell Inn, Tushingham, Malpas (Nov. 14).

Derbys. Muse Lane Farm, Sudbury (Nov. 14).

Durham. Howden Farm, Blakeston Lane, Norton-on-Tees, Stockton-on-Tees (Nov. 9); The Cottage, Hurworth Burn, Wingate (Nov. 10).

Lancs. Rossall Grange Farm, Fleetwood (Nov. 8); Worlds End Piggeries, Benny Lane, Droylsden, Manchester (Nov. 8); 67 Farm Road, Thornton, Blackpool (Nov. 11).

Lincs. Red Berry House, Leadenham (Nov. 9).

Merioneth. Pandy Fields, Dolgellau (Nov. 14).

Norfolk. Hall Farm, Stalham, Norwich; Chapel Farm, Croxton, Thetford (Nov. 9); Primrose Farm, Upton, Norwich (Nov. 11); The Farm, West Caister (Nov. 14).

Notts. Piggeries, Glebe Farm, Clipston on the Wolds (Nov. 9).

Suffolk. Shelland Hall, Bury St. Edmunds (Nov. 11).

Yorks. Swalefield Farm, Morton-on-Swale, Northallerton; Sough Hill, Caldwell, Richmond (Nov. 8); Knabs Pig Farm, Masham Road, Bedale (Nov. 9); Piggeries adjoining Skelton Grange Farm, Leeds; Micklebrack Farm, Leeming Bar, Northallerton; Church Farm, Appleton, Wiske, Northallerton (Nov. 10); Holgate Home, St. Barnabas Road, Middlesbrough; Greenas Farm, Azerley, Ripon (Nov. 11); Horbers Farm, Burton Leonard, Harrogate; St. Trinians, Easby, Richmond; Langwith Farm, Well, Bedale (Nov. 14).

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN
Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 31st October, 1960		8	—	2	94	—	73
Corresponding period in	1959	12	—	—	118	—	75
	1958	5	—	1	33	—	43
	1957	7	—	23	76	—	45
1st January to 31st October, 1960		169	1	30	1,590	—	1,002
Corresponding period in	1959	208	2	9	728	—	1,058
	1958	137	5	116	617	—	1,069
	1957	277	7	117	744	—	809

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

SCIENTIFIC

Teladorsagia davtiani Andreeva and Satubaldin, 1954, in British Sheep

Sir,—The stomach worm *Teladorsagia davtiani* was recently recovered from several lambs which had died from suspected parasitic gastro-enteritis, and had been sent to Weybridge for *post-mortem* examination from farms in the S.E. of England. So far as is known, this stomach worm has not been recorded previously from British sheep.

All of the lambs had mixed infestations. In addition to *T. davtiani,* which was present in appreciable numbers, *Ostertagia trifurcata, O. circumcincta* and *Trichostrongylus axei* were recovered from the abomasum, and *T. vitrinus* from the small intestine.

The male worms of *T. davtiani* and *O. trifurcata* are very much alike. Their spicules can, for all practical purposes, be regard as identical, so that when examined under a low power magnification, it is not easy to differentiate these two species. On careful examination, however, the diagnostic characteristics of the genus *Teladorsagia,* which are, bursa without an accessory bursal membrane, powerful supportive apparatus to the genital cone and sessile papillae at the distal end of the genital cone, can be distinguished and serve to differentiate *T. davtiani* from *O. trifurcata.*

Yours faithfully,
J. H. ROSE.

Central Veterinary Laboratory,
New Haw,
Weybridge,
Surrey.
November 11th, 1960.

The Use of Teoquil for the Treatment of Ringworm in Cattle

Sir,—During the winter of 1959/60 and the following spring, we had an opportunity of investigating an outbreak of bovine ringworm and its subsequent development on a large co-operative dairy farm and the associated calf rearing farm. All the adult animals were of the Friesian (Holstein) breed and had been imported into Yugoslavia during the previous 2 years. The majority of the calves were of the same breed, from these cows, together with a smaller number of calves of the Simentahl breed which had come from privately owned farms. It appeared that the infection had entered the premises through these latter calves. One hundred and twenty-five cases of ringworm were identified and treated. One farm worker was also infected.

When the outbreak was discovered during the winter, all the animals were housed. The housing was satisfactory with the exception of a few of the calf houses where the numbers of animals were too large for the ventilation and cleaning facilities. All infected animals were being fed the same ration, namely a maize silage together with hay and concentrates. The majority of cases were observed in young animals, *i.e.* calves and heifers up to 7 months old. When the veterinary diagnosis was made, it was reported that the disease had been spreading in the animals for several weeks.

The location of the lesions on the milking cow was mainly on the skin of the chest and flanks, rarely on the head or neck, and occasionally on the limbs. In the calves and young heifers the lesions appeared mostly on the head and neck. These lesions were characteristic of the disease, namely clearly marked areas, often circular, with the hairs either broken or scarce and the underlying skin covered with grey scales. In the calves the typical thick-layered crusts were more usually seen, and underlying skin was moist, frequently with bleeding. There were some animals in which only after extremely careful examination could one find one or two infected spots, while on the others the skin was much more affected. In these latter cases the lesions were frequently confluent, and this was seen to be particularly common in calves. It was also noticed on the unpigmented areas of the skin (particularly on the limbs of the dairy cows) that the ringworm lesions also showed erythematous rings.

Material for examination was taken in the usual way by removing broken hairs from the edges of the lesions or by scraping the skin with a sharp surgical spoon. Microscopic examination revealed an ectothrix type of fungus with chains of spherical, large spores surrounding the hair shafts. On culture on Sabouraud's glucose agar, *Trichophyton verrucosum,* var. *album* and *discoides,* was isolated. By using this culture, a biological test was carried out with guinea-pigs. Guinea-pigs were also infected with a culture of *Trychophyton mentagrophytes* for purposes of comparison. In both cases where the cultures had been rubbed into the skin, typical lesions developed.

Following the observations of Crawshaw (1957), Forster (1957), Fowler (1957) and Gold and Jones (1958), we investigated as a treatment a 0.25 per cent. solution of Hexadecamethylene-1:16-bis (isoquinolinium chloride), (Teoquil)*. First we tested the material on the guinea-pig infections described above. The lesions were painted with the undiluted material for 2 or 3 applications. In all affected animals development of the lesions was arrested after the first application and the crusts fell off. In the untreated control animals the lesions continued with a progressive development. Following the application of Teoquil no fungi could be found in material removed either 24 or 48 hours after treatment. Fungal elements could not be isolated on culture from treated cases. It was noted that the treatment was equally efficient in curing experimental infections due to either *T. verrucosum* or *T. mentagrophytes.*

* Tinevet, Allen & Hanburys Limited, London.

In all, the commercial Teoquil preparation was applied to 125 infected animals. Before treatment the crusts were removed from the skins of the calves using a soft brush and water containing some washing soda. When the affected areas were dried the lesions were painted with the undiluted solution. In the case of most of the adult cows the crusts on the skin were fairly thin and the preparation was applied without any previous washing or cleansing. Every affected animal was re-treated either 2 or 3 times at 48-hour intervals. Following the conclusion of the treatment course, the skin was found to be free of crusts in all the treated areas. The epidermis was dry and the epithelium was showing signs of rapid regeneration. Examination of hairs and scrapings removed from the treated lesions failed to reveal any fungi. Cultural examination failed to isolate any fungi from this material.

In conclusion it can be said that the treatment using topically applied Teoquil was completely successful, the average treatment course of an infected animal lasting 4 to 6 days. As far as could be ascertained there were no relapses. A few instances were seen where lesions were found, which due to their situation had not been treated with the others. In these few cases, however, the infection was quickly cured by treatment applied in the same manner as to the other areas. The results obtained from this investigation would appear to confirm the observations made by other workers.

We would like to acknowledge the supplies of 0.25 per cent. Hexadecamethylene-1:16-bis (isoquinolinium chloride) (Tinevet) produced by Allen & Hanburys Limited, London, and kindly supplied to us by Farmitalia, S. A. Farmaceutici Italia, Milan, Italy.

Yours faithfully,
S. FORENBACHER,
M. HAJSIG,
V. SERTIC.

Veterinary Faculty,
University of Zagreb,
Zagreb,
Yugoslavia.

October 27th, 1960.

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GENERAL

Surgical Interference in Dilatation of the Stomach of the Dog (Torsion of the Stomach)

Sir,—I was very interested to read the article under the above heading by J. B. Tutt and T. G. Yarrow, in the November 12th issue of THE VETERINARY RECORD. Previously I had not read of any recorded successes in this condition, in THE VETERINARY RECORD.

Perhaps quite a number of members have been fortunate enough to encounter the condition in its reasonably early stages and been able to operate with success. That may be the case, but Messrs. Tutt and Yarrow are to be congratulated, inasmuch as they took the trouble to record the case in your columns.

I have seen 5 such cases in the past 9 years. The first 3 were *in extremis* by the time I saw them.

About 2 years ago I operated on a Boxer bitch, which had developed the condition suddenly, one Bank Holiday afternoon. By the time I saw the bitch, about 3 hours had elapsed and she was stretched out, grossly distended and acutely shocked. I expected her to die in transit to the surgery, but she survived, to receive Nembutal anaesthesia and laparotomy.

Foolishly, the torsion was relieved before relieving the acute gaseous pressure in the stomach, and quite naturally a mixture of fluid and gas shot up into the pharynx, and of course some was inhaled. I think the bitch was too far gone in any case, but this mishap caused me to destroy her at this juncture.

About 6 months ago a Springer Spaniel dog, aged about 10, was brought by car to the surgery, with the story that he had jumped over a wall about an hour previously, and since then had made repeated abortive attempts to vomit, and had progressively become greatly distended and was quite distressed. Surgery was carried out about an hour later, under Largactil and Nembutal anaesthesia. A medium-bore intravenous needle was attached to a flutter-valve, and used to release the gas and allow it to escape well away from the patient. In this way, the stomach soon reached more reasonable proportions.

I fully agree with Messrs. Tutt and Yarrow regarding the difficulty of describing the re-positioning of the spleen and the stomach. It was trial and error until one felt it looked correct.

Perhaps it was because of this inability to understand exactly what I had done—plus laziness—that I did not venture to record the case at the time.

The dog recovered completely, having received routine penicillin/streptomycin for 3 days.

I had not found it necessary to incise the stomach and remove contents in my case, though perhaps by not doing so one may be running the risk of regurgitation. Also, I had not anchored the stomach, and there was no recurrence of the condition. Perhaps the chance of a repetition of the condition is negligible (as in contrast to abomasal displacement), but anchoring is certainly very logical.

I am sure it would be helpful to many of us to hear, through THE VETERINARY RECORD, of any similar cases, and in what respects techniques were found to be faulty or successful.

Yours faithfully,
C. STEELE.

65 Seaview Road,
Wallasey,
Cheshire.

November 14th, 1960.

Tumours of Farm Animals

Sir,—I found Dr. Cotchin's paper very interesting, particularly his reference to tumours in young animals.

Two cases recently encountered here may be of some interest.

Case 1:

A 2-month-old Hereford calf was missing 3 days, and when found could stand only when supported. Two days later it was unable to stand at all, although it could eat and drink, and reflexes appeared to be normal. It was destroyed 5 days after being found.

On *post-mortem* examination the cranial dura mater showed 5 raised circular firm glistening areas up to the size of a sixpence in diameter. These were causing indentations on the left cerebral hemisphere.

Laboratory diagnosis was " malignant lymphoma."

Case 2:

An 8-month-old Shorthorn cross heifer had difficulty in breathing and swallowing, and a grossly enlarged larynx was palpable both externally and internally.

Post-mortem examination showed the swollen larynx to be almost occluded, and the oesophagus was also partially obstructed. The laryngeal tissue was very firm and resistant to cutting. There was some metostasis to adjacent lymph glands.

Laboratory diagnosis in this case was " fibrosarcoma."

Histopathology was carried out at the veterinary laboratory of the Alberta Department of Agriculture.

Yours faithfully,
Wm. C. McNISH.

Box 820,
Lloydminster,
Alberta,
Canada.
October 31*st*, 1960.

Infectious Equine Coughing

Sir,—To those inquisitive of further details on this subject may I refer them to the report of O. Waldmann and K. Köbe on this condition, a translation of which appeared in THE VETERINARY RECORD during 1936 (*Vet. Rec.* **48.** 80).

" The great J. T.," who was closely associated with THE RECORD in those days, considered this report of such importance that it should not be dealt with merely in the form of an abstract, but that a full translation of it should be published. No doubt in this " virus age " further research has been done, or should have been done, but this report still is very interesting reading.

I had the good fortune to meet on several occasions J. Pape, official—if I may use that term—veterinary surgeon at Hoppegarten, Germany's Newmarket, who himself had done much work on this subject. If my memory has not failed me too greatly, he stoutly maintained that a period of high temperature always occurred in the early stages of infection, but that it was of such short duration it was often missed; and further, that if unfortunately a horse should be raced, or subjected to serious training work during this period then it was almost certain to develop pneumonia. Hence the great necessity for the free use of a thermometer in a stable and the strict avoidance of serious work in animals affected.

It was worthy of notice, in the report referred to, that these workers concluded that this virus disease of horses was transmissible to pigs and cattle, and it would be of considerable interest to know whether this has been observed during the present outbreak in this country.

Since it is more than likely that many will find it somewhat difficult to read the publication named, you, Mr. Editor, may consider whether, after this quarter-of-a-century lapse of time, it is allowable and of sufficient interest to re-publish Part X.—Summary of this Report.

Yours faithfully,
E. BRAYLEY REYNOLDS.

Newmarket.
November 7th, 1960.

The summary was as follows:—

(1) Epizootic cough in horses is caused by a filtrable virus. All other breeds of horses as well as blood-stock are equally susceptible to infection.

(2) The virus infection sets up lesions of bronchitis and peribronchitis.

(3) The chief symptom of the disease is a dry, deep cough.

(4) The disease is readily transmissible from horse to horse.

(5) The incubation period is usually from 2 to 3 days.

(6) After infection with the virus, the animal becomes immune. The degree of immunity seems to depend upon the severity of the attack.

(7) When an affected animal is retained in unfavourable environmental conditions the attack brought about by the virus alone may be complicated by a secondary infection, which is produced for the most part by *Diplococcus pneumoniæ* (Shutz, 1887).

(8) Secondary infection with bacterial invaders confers upon an outbreak the clinical characteristics of an infectious bronchopneumonia.

(9) Epizootic cough in horses is identical with Hoppegarten cough, epizootic laryngotracheal catarrh, infectious catarrh of the air passages, and skalma, and if complications set in caused by secondary bacterial invaders, with Brussels disease (" shipping fever ") or infectious bronchopneumonia.

(10) The virus of epizootic cough is transmissible to pigs and cattle.

(11) Infectious bronchitis of cattle is apparently also caused by the virus of epizootic cough in horses.